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AN ELECTRICAL THEORY OF NEURAL ENERGY*

CHARLES K. MILLS, M.D., LL.D.

PHILADELPHIA

AN EXAMPLE OF THE COMPLEXITY AND RAPIDITY OF NERVOUS ACTIONS

In the "Memoirs and Letters of Sir James Paget" by Stephen Paget, one of his sons, is related a remarkable example of the complexity and rapidity of the transmission of nervous impulses. At the request of Sir James, Mademoiselle Janotha a celebrated pianist played on the piano a presto by Mendelssohn, one of the swiftest compositions known to her. The time was taken, and the number of notes was counted. Altogether she played 5,995 notes in four minutes and three seconds, rather more than twenty-four notes a second. Sir James calculated that there were at least no less than seventy-two distinct variations in the nervous impulses transmitted from the brain to the muscles in each second. He also estimated that there were at least five distinct and designed qualities in each of the seventy-two movements. In addition, there were at least four conscious sensations for each of the twenty-four notes in each second.

> That is, there were at the rate of ninety-six transmissions of force from the ends of nerve-fibers, along their course to the brain, in each of the same seconds during which there were seventy-two transmissions going out from the brain along other nerve-fibers to the muscles. And then add to all this, that during the time, in each second of which the mind was conscious of at least ninety-six sensations, and directed not less than seventy-two movements, it was also remembering each note to be played in its due time and place, and was exercised, with a judgment, in the comparison of the playing of this evening with those of the time before, and with some of the sentiments which the music was intended to express.

Other factors besides those of memory entered into the performance. The pianist was sure that she could have played from notes as easily as from memory, and this would have added sight to the four sensations associated with each note.

Even if these calculations are not exactly correct, they are sufficiently so to show the wonderful manner in which nervous impulses are con-

* Read before the Philadelphia Neurological Society, May 16, 1924.

veyed to, through and from the brain in a single second. By such an observation as this, although not timed by the means employed by the physiologist in his laboratory, we are able to arrive at an idea of the complexity and rapidity of neural activity better than by the usual methods of the laboratory. Such laboratory research, however, is by no means vain nor valueless, although necessarily wanting in some respects.

THE NEURAL STRUCTURES CONCERNED WITH THE CONDUCTION OF NERVOUS IMPULSES

In order to arrive at some idea of neural energy and conduction in the nervous system, we must first have a clear view of the structures concerned with the function of conduction. The older views were comparatively crude and coarse. They recognized only the bundles or cables of peripheral fibers called nerves, and with regard to the central nervous system, illy defined or undefined tracts proceeding from sensory nerves through the spinal cord into the brain and tracts equally illy defined going from the brain centers through the spinal paths and to muscles, glands and other peripheral tissues or organs. Then arose the neuronal theory which grew out of the fine work based on the Nissl stain. Nissl and Cajal held first place in this work, and Waldeyer eventually clarified the views on the subject by his theory of the neuron.

The neuron did not represent the ultimate dissection of nervous structure, and soon we were confronted with the neurofibrillary theory. Even before the adoption of the neuron theory, observations were made which pointed to the fibrillary structure of the nerves and of the central nervous system. Max Schultze, as early as 1872, observed the existence of fibrils both in axons and cell bodies, and later a few similar observations were made by others, but it was not until the time of Apathy that a neurofibrillary theory began to hold a definite place in neuro-anatomy and neurophysiology.

The neurofibrillary theory yielded steadily increasing evidence that neurofibrils exist that are much finer in their structure than axons and dendrites. It even became probable that these neurobrils are themselves made up of still more elementary fibrils. It also became probable that in a given neuron the total number of primitive fibrils equals the total number of endings of both axons and collaterals. It was found by Apathy, Bethe and others that fibrils of varying sizes were present not only in the cell body and its processes, but also in large numbers in the spaces between the cells; in other words, the neurofibrils were intracellular and intercellular.

At the meeting of the Academy of Natural Sciences, March 4, 1902, I presented a verbal communication entitled, "The Neurofibrillary Theory and Its Bearings upon the Localization of Function in the

Nervous System."[1] Referring to the correlation of nerve energy with the other forms of physical energy, I held that the elementary fibrils concerned with neural energy not only conduct or transmit nervous impulses, but also, by means of the special manner in which they are arranged in the nerve centers as well as in the periphery, determine the intensity and character of the discharge. I suggested that the fibrillary coils and bundles in the central nervous system represented a complicated induction apparatus. I held that localization of function is brought about by means of special arrangements of intracellular and intercellular neurofibrillary coils and plexuses in the particular regions of the brain called centers.

Later I published a fuller consideration of this neurofibrillary theory, under the title of "The Neuron and Neurofibrillary Theories of the Nerve Cell with Some Consideration of Neural Energy and Neural Mechanisms."[2]

In this communication, I tried to show the manner in which separate neurons or nerve cells were held together by uniting neurofibrils; how these neurofibrils entered and left the cell bodies; how they were collected in coils or spirals both in cell bodies and in the interspaces between these cell bodies; how they were distributed in the axons and in their collaterals and terminal arborizations; how neurofibrils concerned with certain special functions sought out, as shown by Wolfstein, the particular regions of the central nervous system to which they were destined; and how, finally, they collected in special functional centers or areas in the brain, these areas representing certain districts in which centralization or polarization of function occurred. I indicated that the whole represented a series or collection of electrical appliances concerned with intensifying and segregating special functions.

ELECTRIC FISHES

The idea that in the central nervous system are collections of nervous matter in the form of coils and spirals of neurofibrils, these being both intercellular and intracellular, which play the part of electric organs, and that these stimulate and more or less control conduction and other neural functions, is borne out by what is known of the electric organs of the various forms of electric fishes, such as the malapterurus, the electric ray, the electric eel and the electric torpedo. Their central electric organs or "plates," as they are called, have been compared by various

1. Mills, C. K.: The Neurofibrillary Theory and Its Bearings Upon the Localization of Function in the Nervous System, Proc. Acad. Nat. Sc. of Philadelphia **56**:113 (March 4) 1902.

2. Mills, C. K.: The Neuron and Neurofibrillary Theories of the Nerve Cell with Some Consideration of Neural Energy and Neural Mechanisms, Philadelphia Hosp. Rep. **6**:31-47, 1905.

authorities to groups of ganglion cells, and the conductors by which the effects of the electric discharges are produced are undoubtedly nerves.

Many experiments have been performed on varieties of electric fishes, such as the gymnotus and the malapterurus, by men of no less authority than Michael Faraday and DuBois-Reymond. Among other experiments, applications of electric currents similar to those used in the well-known nerve-muscle experiments on the frog have been made, and with similar results. Not only do the well-known electric fishes possess the power of electric discharge, but the same property is possessed in less degree by many other fishes, even by the common eel.

NEUROBIOTAXIS

The principle of neurobiotaxis, which has been especially discussed by Ramon y Cajal and Ariëns Kappers, has something to do with the property of neural conduction. The facts have been arrived at by microscopic study of the cells of embryos.

Cajal believed that the connection of nervous elements is determined by the secretion of "attracting and repulsing substances and by the sensibility of these substances in the ganglion cells."

In examining the cells of embryos, Kappers found that they have an unusual tendency to shift. This shifting, he believes, is due to a bio-electric act. It is in obedience to this same bio-electric action that offshoots or processes develop as axons and dendrites. Conductivity like growth follows the line of least resistance, and both are bio-electric processes.

The broad general idea evolved is that during evolution of tissue, electric conditions occur which are very similar to those which take place in stimulation, and thus connections are made between the nerve fibers and the parts they activate.

EXPERIMENTS TO DETERMINE THE RATE OF CONDUCTION OF NERVOUS IMPULSES

Since the middle of the last century, many physiologists have made efforts to determine the rate at which nervous impulses pass in the peripheral nervous system, hoping thereby also to determine the rate of conduction in the entire nervous system, peripheral and central. The textbooks of physiology, like that of Ladd and Woodworth,[3] contain sufficient accounts of these efforts, making it unnecessary for me to go into this subject at any length.

Some especially interesting experiments and observations have been made in recent years, as those of Keith Lucas[4] and his collaborator

3. Ladd, G. T., and Woodworth, R. S.: Elements of Physiological Psychology, New York, Charles Scribner's Sons, 1911.

4. Lucas, Keith: The Conduction of the Nervous Impulse, A Monograph on Physiology, rev. by E. D. Adrian, New York, Longmans, Green & Co., 1917.

E. D. Adrian. This monograph was the outcome of lectures delivered at University College, London, by Keith Lucas just at the outbreak of the World War.

Two theories regarding the conduction of nervous impulse have chiefly held the field, namely, the chemical and the electrical.

By the way, members of the Society will recall that Dr. Weir Mitchell, for several years our own president, made neural energy the chief topic of his presidential address before the American Neurological Association in 1909. He adopted with some reservations the chemical theory, giving the usual arguments adduced in favor of this theory.

The chief objection to the chemical theory of nerve conduction is founded on the fact that the conduction is not accompanied by true catabolic manifestation, by mechanical, chemical or thermal changes, or these are so slight as not to be experimentally discernible. Many experiments on nerve fatigue have been performed, and they also contradict the chemical theory. It has been shown that a nerve can be indefinitely submitted to the action of a faradic current without any resulting fatigue, as demonstrated by subsequent examinations of the nerve or subsequent use of it. A nerve placed under the influence of curare can be stimulated indefinitely by electric currents, and after the withdrawal of the influence of the drug will be found unchanged in its power of conduction. The amount of work that the nerve can do without permanent fatigue is remarkable.

The rate at which a nerve can conduct has been studied by means of an electric current by many experimenters since DuBois-Reymond. Helmholtz made other experiments before the middle of the last century. He was among the first, if not the first, to determine the rate of conduction in the motor nerve by the methods still used, although with some modification and variation by physiologists. This was simply to expose the nerve connected with the muscle—the nerve and muscle of a frog, for instance—to an electric current and note its rate through a certain length of nerve by means of a galvanometer. Helmholtz arrived at a conclusion not very different from that held by physiologists of today, that the rate of conduction in the peripheral motor nerve was about 100 feet a second, while the rates in the sensory nerves were a little less. It is not all of the problem, however, to obtain a result of this kind in this way, and one is sometimes inclined to half agree with Johannes Müller. In a discussion on this subject with Helmholtz, Müller remarked that the rate of conduction could not be determined, central or peripheral, as he thought it was the same as that of light.

At any rate, it is my opinion, as indicated in the story of Paget already outlined, that the rate of nerve conduction is much greater than that usually assigned to it by experimenters on peripheral nerves.

In trying to arrive at the rate of conduction in peripheral nerves, physiologists have obtained many striking results by means of experiments with rapidly interrupted faradic currents. They have made it clear that a nerve has a certain refractory or subrefractory stage; that its conductivity can be decreased by cold, and increased by heat; that such increase or decrease can be brought about by variously using electric currents as regards their intensity and rapidity of vibration; that by submitting the nerve to various substances, such as alcohol, carbon dioxid and narcotic drugs, the rate of conduction may be decreased in the part of the nerve submitted to these agents, although its power of conduction is not destroyed.

These and many other experimental facts have seemed to indicate that nerve conduction is electrical rather than chemical. In experiments similar to those with "core models" the nerves are placed under the influences of certain solutions, and the resulting electric currents are studied.

Every clinical neurologist in the practice of his specialty frequently performs experiments in nerve conduction as, for instance, when he examines to determine the amount of retardation of pain, heat, cold, touch or any other phase of sensation, or when he irritates a certain peripheral nerve to produce any of these sensations.

One is also studying rates of conduction when, in operating on the brain, one applies bipolar or unipolar electrodes carrying very weak currents to the motor region of the cerebrum while searching for the proper place to excise a piece of the cortex in order to treat a patient who has epilepsy, with unilateral or monolateral symptoms. The only trouble in such a case as this is that the experimenter is not justified in repeating the experiments more than is necessary to obtain the point of excision. My observation in such a case is that, as near as one can determine, the rate of conduction from the brain to the muscular periphery is practically instantaneous.

Exner says, as shown by Lorbeer:[5]

> Nerve stimuli applied together in point of time to the fore limb region of a rabbit's cortex and to the skin of the crossed foot exert a facilitating influence on each other. Again, a sound conveyed to the ear of a chloralized rabbit increases the amplitude of a reflex movement of the foot, induced by a stimulus applied to the foot a moment later.

The well-known facts of electrotonus are in favor of the electrical theory of nerve conduction, as are other well-known facts such as the occurrence of phosphenes from irritation of the retina by light or from

5. Lorbeer, Floyd Irving: Introduction to the Philosophy of Mind, in the Electrical Nature of Nerve Conduction, p. 11.

a blow on the head. Symptoms like those of waking numbness and many others might also be adduced in favor of the electrical theory.

AN ELECTRONIC THEORY OF NERVOUS CONDUCTION AND NEURAL ENERGY

When the atom was supposed to be the smallest indivisible form of matter, our hypotheses regarding nerve conduction and neural energy in general were necessarily atomic. In other words, the hypotheses involve the question of what happens to these atoms when neural energy in any form is exhibited. It was known that certain chemical changes took place in these atoms under the influence of neural work, and hence it was natural to believe that nerve conduction was a chemical process. I have shown, however, that numerous reasons based on experiments might be adduced to disprove these hypotheses, mainly that nerve conduction is unaccompanied by the evidence of metabolism that can be easily demonstrated to be present as the result of non-neural work.

The present conception of matter is that an atom is not indivisible, but is made up of really indivisible material called electrons. The number of electrons in an atom differs, according to the particular elements, from one or two in hydrogen or helium to many scores in elements like uranium. The definition of an electron, like that of electricity itself, seems to be somewhat uncertain. One knows that electricity is present from the behavior of bodies that are supposed to be electrified, and one knows, in other words, that electricity is the property exhibited by matter under various circumstances.

Merely to assert that electricity is a force like light does not much help our comprehension of its phenomena; to view it as an effect or strain of an "imponderable ether" is not much more helpful. In these later days, the "ether," like many other conceptions long held, has lost its status, and one finds it easier to conceive of something real, however minute, which we call electrons, filling all space and penetrating all matter. This is one of the views, or one of the outcomes of Einstein's views, that will be further presented by Dr. Dercum.

Personally, I think of an electron, however minute, as something real, something material, although its actual size is described as being about one hundred-thousandth of the dimensions of its orbit, this orbit being so minute that it is measured in millionths. It is not more difficult for me to imagine this exceedingly minute bit of matter than it is to comprehend the little organisms that are seen in apparently pure water with a magnifying glass, or germs like the tubercle bacillus or the spirochete of syphilis or other microscopic organisms that can be seen by higher and higher to the highest visible magnification. On the other hand, is it not more difficult to comprehend such a Lilliputian body than

it is to comprehend the distances and dimensions of bodies in the universe whose light requires hundreds or thousands of years to reach the earth.

Many of the accepted observations on the electrons and hydrogen nuclei are based on certain facts. A study with the spectroscope, for instance, reveals optic spectra or roentgen-ray spectra, the character and significances of which have been determined. The electrons of one element can be used, as they were used by Rutherford,[6] to bombard and thereby change the character of another element.

Therefore I conclude that electrons, whether filling space and exhibiting themselves by streams of light, or whether occupying minute neurofibrillary tubes and in some way conveying nervous impulses, are real. One cannot have an electrical view of nervous conduction without taking the electrons into account. The speed of conduction is not far removed from what mathematical determination has inferred that electronic speed may be.

In talking over the subject of electrons with my friend, Dr. Lloyd, he said, "Did you ever see an electron?" I might with equal propriety have asked him, whether he had ever seen a "thought," or the so-called "imponderable ether." If we can only reason on what we see or touch, many of the truths of science and philosophy will have to be discarded.

METHOD IN WHICH ELECTRICAL CONDUCTION TAKES PLACE IN THE NERVE

While I believe that conduction of nervous impulses is an electric process, it is somewhat difficult to explain this. Supposing that my theory is correct that neuropils concerned with the development or concentration of electricity are present in the brain and at other levels of the cerebrospinal system, in what manner are the impulses transmitted through this system? In other words, how do they pass from the motor centers of the brain through the cerebral tracts to the basal and spinal centers, and thence through the peripheral motor nerve to the muscle? It is not a process in which one electron hits another and this another, like the bumping procedure in an Oxford boat race.

Probably the views of Ralph S. Lillie[7] give a clue to the manner in which the impulses are conveyed. In a paper, he discussed this subject at length. He compared nerve conduction with conduction in iron, and the changes which are produced in the latter by acting on it with nitric acid with the changes that occur in nerves in the process of transmission. In some of Lillie's papers, he discusses in detail the "comparison between

6. Rutherford, cited by Russel, B.: "The A B C of Atoms, E. P. Dutton & Co., 1923.

7. Lillie, Ralph S.: Transmission of Physiological Influence in Protoplasmic Systems, Especially Nerve, Physiological Rev. **1**, No. 1 (Jan.) 1922.

the phenomena of activation, transmission and recovery in the living system and in the passive iron model."

The propagation of impulses in the nerve is due, according to Lillie, to the effects of locally produced currents rapidly succeeding each other in the nerve. These local currents are similar to those produced between the films which form on the surface of iron and the face of the iron itself. It must be understood that these currents succeed each other with great rapidity in the process of propagation of nervous impulses. Given the central neuropil, the electronic power that is started at a central point will pass through the nerve tracts, producing rapidly succeeding local currents, the whole constituting the entire transmission through tracts and nerves, or through nerves and tracts.

NEURAL ENERGY AND THE MIND

This article has been concerned chiefly with the cerebral and other structures that take part in the initiation and especially in the conduction of nervous impulses. Little has been said about the mooted question of the material or immaterial theory of the mind. It may be true, as Lorbeer suggests, that mental activity conforms to electrical principles, but this does not mean that mind and electricity are absolutely identical, but only that mental activity is correlated with other great forces like electricity and light. Mind is the resultant of the numerous and complex activities of the brain. Impressions from without pass to the brain by a process of electrical conduction. The centers of the brain are associated in a wonderful complex by processes that are in accordance with electrical principles. No mentality is possible without structural and functional cerebral association. Mind, brain and outside natural forces are correlated. Neural energy is most philosophically expressed in terms of electricity; it is electronic, according to the views of the latest physicists.

THE NERVOUS SYSTEM IN THE LIGHT OF EINSTEIN'S INTERPRETATION OF ENERGY

WITH A BRIEF CONSIDERATION OF THE DOCTRINE OF RELATIVITY *

FRANCIS X. DERCUM, M.D.
PHILADELPHIA

The temptation to extend physical and mathematical conceptions into the domain of living forms is almost irresistible. On all sides, however, we are confronted by problems that contain large numbers of unknown factors, and yet had Einstein hesitated for this reason, the remarkable results which he has achieved would never have seen the light of day. In making a plunge into the unknown, too, we are comforted and encouraged by what Huxley has said in regard to the scientific imagination, an imagination which is so often a forerunner of scientific progress.

I will begin by making a brief statement, stripped of its mathematical formulae, of the basic features of Einstein's doctrine of relativity.[1]

First, a given object is definitely related to the three classical dimensions of space; i. e., to three planes placed at right angles with each other. The position of such an object can always be determined by measurements made from these planes.

Second, every object in the universe is moving; the object which is the subject of our measurement is therefore moving, and "time" is required for it to move from one point to another. Time therefore enters at once into our conceptions of space relations and becomes of necessity a fourth measurable factor. To the three static dimensions of space, then, we are obliged to add a fourth dimension, that of time dependent on motion, and space therefore becomes, so to speak, four dimensional; it reveals itself to be a "space-time continuum."

Third, according to the Newtonian principle, a body persists in a state of uniform motion in a straight line unless it is compelled to change that state by forces impressed from without. As the number of

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* Read at a Special Meeting of the Philadelphia Neurological Society, May 16, 1924.

1. This statement, presented in a most elementary form, is the interpretation derived from a consideration of the writings of Einstein; from Malcolm Bird's compilation of Einstein's theories based on the essays submitted in the competition for the Eugene Higgins prize and published by the Scientific American Publishing Co., New York, 1922; from James Hopwood Jeans' admirable presentation of the subject in the Encyclopaedia Britannica, New Volumes **32**:261, 1922, and from other sources.

bodies in the universe is multiple, a given body necessarily comes within the gravitational fields of other bodies. If it comes within the gravitational field of another body, it cannot continue to move in a straight line, but its motion of necessity becomes curvilinear.

Let us consider some of the elemental facts of our abstract modes of thinking. A point as such is an abstract conception. A line consists of an infinite number or points arranged in sequence. A line, also, in this sense, is an abstract conception. A plane consists of an infinite number of points arranged in the relation of two dimensions; also an abstract conception. As soon as the conception of a third dimension is added, it ceases to be a plane. I can move my hand up and down, to and fro and from side to side, but there are neither lines nor planes in the outside world to correspond to these motions. They are only abstractions in my own mind. It takes time to make these motions, and the only factors of the reality of which I have convincing proof are the two facts of space and time, and these, as we have just seen, are in nature inseparable. That which is about me is the space-time continuum. It is the only objective reality. All else is abstraction. It alone is based on experience.

Further, the term space-time continuum merely expresses the fact of moving matter. All space is filled with moving matter; nay, all space *is* moving matter. All space is coexistent and coeval with the space-time continuum, i. e., with matter in motion. If there is no matter, there is no space. If there be no space, there is nothingness, and nothingness has no place in our problem. When Einstein speaks of the curvature of space, he speaks of the curvature of matter or rather of the curvature of the space-time continuum.

Euclid's interpretation begins and deals with fixed points, lines and planes. Newton's interpretation begins with a body at rest or, at most, with a body moving in a straight line. No such things as fixed points, lines or planes exist in nature; no such thing as a body at rest or a body moving in a straight line exists in nature. Both interpretations necessitate conceptions which—to borrow a phrase from Herbert Spencer—are illegitimate. Both necessitate mental pictures which have no counterpart in reality. Einstein, on the other hand, starts with the universe as it actually exists—with matter moving in relation with space and time. In other words, he begins with the ever moving, ever changing space-time continuum, a continuum in which we are immersed and of which we form a part.

The difference between the abstract conceptions of the plane geometry of Euclid and the actual findings in nature is at once obvious. While the theorems of Euclid are of course true in the abstract, they are only partly or relatively true in their concrete application, and we

are not surprised to find that the shortest distance between two points is not always a straight line, that the sum of the angles of a triangle is not always 180 degrees or that the angles of a square are not always 90 degrees. Euclidian geometry is of necessity static; Einstein's conception is of a moving, a living, a dynamic reality.

THE EINSTEIN DEMONSTRATION

The foregoing considerations of the structure of the space-time continuum led Einstein to a remarkable and startling inference. If light is an electromagnetic phenomenon, if it consists of electrons in motion—in other words, if light is made up of *matter* in motion—it must have weight, and if a beam of light should come within a gravitational field, it must sag like a string or a telegraph wire. This inference was expressed mathematically and with great accuracy by Einstein, and it was verified in a brilliant manner by the Royal Society and the Royal Astronomical Society. Two expeditions were sent out by these societies, one to Sobral (Brazil) and the other to the Island of Principe (West Africa), made up of some of Great Britain's most celebrated astronomers. They observed the solar eclipse of May 29, 1919, and their results confirmed Einstein's theory in a most satisfactory manner. More recently, the truth of Einstein's theory was definitely demonstrated by the observations made at Wallal in northwestern Australia, by Campbell in the solar eclipse of 1922 and reported by him to the American Philosophical Society in 1923. These observations were confirmed by studies of the photographic plates by Trumpler and reported by Campbell to the American Philosophical Society in 1924.

SPHERICAL CONTINUUM

Now let us introduce into our conception instead of points, lines and planes, the surface of a sphere. The lines that represent a meridian or the equator, when followed necessarily return on themselves; they are circles; there is neither a beginning nor an ending. Second, let us add to the conception of meridians and equator the conception of planes perpendicular to these lines, i. e., fulfil the conditions of a three dimensional space. Now just as in fact all lines become curved, so do these planes become curved, i. e., they eventually curve on themselves; they partake of the principle of sphericity, and from this point of view, the universe becomes a "spherical continuum." Thus abstract reasoning confirms the conclusion as to the general curvature of matter, i. e., of the moving matter that constitutes the space-time continuum. It may here be added that this conception of a "spherical continuum" gives rise, among other things, to the inference that the universe is neither finite nor infinite; like the circle, it has no beginning and no ending.

SPEED AND DIRECTION OF MOTION

Finally, a body traveling through the space-time continuum travels in relation to the four dimensions. Its course is spoken of as a "world line" or a "geodesic." A geodesic is the curve of smallest length. It is the analogue of the hypothetical straight line of plane geometry.

Bodies do not move, as we have seen, in straight lines; their speed and direction of motion are constantly changing. To these changes, the term "acceleration" is applied, which means either that the speed is increasing or diminishing or that its direction of motion is changing, or both. Newton began with a body at rest or pursuing a uniform motion in a straight line; Einstein began with the world line of the four dimensions and deduced therefrom the observed facts of gravitation. The world lines, occupying the four dimensions of space, consist necessarily of space-time combinations. They are strained and distorted, owing to the attraction that bodies exhibit for one another. In other words, the phenomena of gravitation express the strain of space-time combinations. The influence of acceleration and the relativity of gravitation are seen in the following familiar illustration. A revolving axis such as is seen in the old-fashioned governor of a steam engine, which has attached to one extremity a rigid though freely movable arm, permits the arm to lie close to the axis when the axis is at rest, but when the axis revolves, the arm is thrown out, and as the speed of the revolution of the axis increases, the arm is thrown farther and farther from the axis. Again, it has been calculated that if the earth's rotation were increased seventeen times, Newton's apple would not fall in a direction toward the earth's center, but would pursue a course parallel to the axis of the earth. If the rotation were increased very much, the apple would pursue a course away from the earth at right angles. The reverse of course obtains with the slowing of the speed of rotation. In either instance, we observe the effects of "acceleration." Again, in Einstein's hypothetical experiment of a man enclosed in a falling box, if the man and box fall at the same rate, the man experiences no sense of weight relative to the box, and he might occupy any position in the box—in contact with the floor or the roof of the box, or not in contact with either. Weight would become manifest only when there was a change in the rate of the fall of the box. Obviously, too, in such a box the pans of a pair of scales would reveal no difference between a pound of lead and a feather. Everything depends on the fact of acceleration.

Further, every particle of matter travels through the space-time continuum in the most direct possible path, i. e., through its world line or geodesic. It would appear, then, that the course of such a particle is dependent on the inherent structure of the space-time continuum.

If this be true, it must explain not only the phenomena of gravitation but all other phenomena as well. Clearly, this principle must apply to all bodies, large or small. It applies to the motions of the planets on the one hand and to the movements of the electrons about the nucleus of the atom on the other. It would seem to apply alike to the coarse mechanical effects of gravitation and to the more recondite phenomena of chemical action, electricity, magnetism and light. It would seem, as regards forces other than gravity, that the latter are merely the outcome of the details of the curvatures, i. e., of the geodesics, traversed by the particles in motion. Such details appear to add merely to the complexities of the problem, not to alter its principle. Such a view has already been advanced by H. Weyl, and his predictions coincide completely with the known facts of the electromagnetic forces. It would appear that all of the expressions of energy are but maelstroms, whirlpools and eddies in the mass of the space-time continuum in which we live.

IMPACT AND TRANSMISSION IN NERVOUS SYSTEM OF ANIMALS

It seems a far cry from the consideration of these problems to the phenomena presented by the nervous system of animals. However, here again problems of motion present themselves, problems of impact, of transmission, of response. To the observer who regards the universe as a whole, the problems presented by the world of living things cannot differ in their essence from those presented by nonliving things. The fundamental principles applicable to one cannot differ from those applicable to the other.

Let us begin with the elementary facts of impact and transmission. The transmission of impacts is illustrated in many ways. It finds its simplest expression in protozoans and sponges. In protozoans, as in the ameba, the transmission of an impact is exceedingly diffuse and slow. This is typically seen in the behavior of an ameba in response to a food particle. It is seen again typically in sponges.[2] Thus if a pin is stuck into a finger of the fresh water sponge Stylatella at 1.5 cm. from the pore opening, this opening—the osculum—will close in about ten minutes. The sluggish transmission on which this reaction depends represents without doubt that elemental property of protoplasmic transmission from which true nervous activity has been evolved. When, however, the syncytic tissue of the epidermal layer of sponges is replaced by tissue in which cellular differentiation has begun and in which special pathways of transmission have begun to be evolved, transmission becomes alike less diffuse and much more rapid. This is

2. Parker, G. A.: The Elementary Nervous System, Philadelphia, J. B. Lippincott Company, p. 26.

seen typically in jelly-fishes and sea-anemones. In the former, impacts on the margin of the bell are diffused through deeper lying contractile cells which form the sphincter-like structure between the centrally located mouth of the animal and edge of the bell. In sea-anemones, an impact on the surface of the animal results in a retraction of the oral disk. Investigations have shown that the impact both in jelly fishes and sea-anemones is diffused through a network. This network, though well defined, is diffuse and continuous, and it also contains cells. The fact that it is diffuse and continuous suggests an analogy with the syncytic tissue of the dermal layer of sponges. Transmission, however, is vastly more rapid in coelenterates than in sponges. The cells that the network contains are the forerunners of the nerve cells of higher animals, and Parker, of Harvard University, has termed them the protoneurons.

We find that the pores of sponges may be closed not only by the extension of a pore membrane, but also by the closure of the canal leading to the pore, the epithelial lining of the pore canal apparently contracting after the manner of a sphincter.

In the jelly-fish and the sea-anemone, the muscle cell no longer receives the impact directly from the environment. There is now interposed an epithelial cell which receives the impact and transmits it to the muscle cell. Later, a third structure appears, which is interposed between the receiving cell and the muscle cell. The function of this new structure—the protoneuron of Parker—appears to be to distribute to the muscle cell or cells the impacts received through the receiving cells.

As already indicated, the nervous system of coelenterates consists of a diffuse and continuous network featured at intervals by cells which are the homologues of the nerve cells of higher animals; in general terms, we deal with a tissue essentially syncytic in structure, for the cellular elements are in no sense separate from each other. No matter how complicated the network may become, its fibers are continuous, and there is free interchange between the cells of the fibers of this network. There is, therefore, a wide diffusion of transmission which is totally different from the transmission along definite paths observed in vertebrates.

In vertebrates, each nerve cell is a separate and distinct histologic integer. By means of its processes, it comes into proximity with other nerve cells, often far distant. The approximated end formations of two nerve cells is spoken of as a synapse. In the nerve net of the coelenterates, transmission of impacts diffuses in all directions; in the neuron of the vertebrate, transmission takes place in one direction only: from the dendrites through the cell body and out through the axon. For instance, while it is possible to elicit a response to a stimulus applied in the course of an afferent neuron of the spinal cord, as in obtaining

a spinal reflex, no amount of stimulus applied to the efferent neuron, for example, to the central end of a divided motor spinal root, will elicit any response.

IONIC THEORY OF STIMULATION AND NERVE CONDUCTION

In considering the nervous system of vertebrates, the question of the neurofibrils of course presents itself. In the earthworm, fibrils pass continuously without interruption through the nerve cells and from cell to cell. Neurofibrils may possibly be found in the neurons of vertebrates, but that they pass from cell to cell through the synapses is not generally conceded. For myself, I adhere to the conception of the neuron as here outlined and to the conception of the synapses. The relations between two nerve cells at the synapses may be ameboid or the synapses may form merely an electro-endosmotic layer. But our conceptions of the structure of the nervous system will not influence the physical principles concerned in its activity. However, to me it does not seem philosophical to interpret the highly differentiated nervous system of the vertebrate in terms of a tissue essentially syncytic in its nature.

About twenty-two years ago, Dr. Mills formulated a theory as to the electrical nature of nervous action. Two years later, Jacques Loeb and Walther Nernst independently concluded that in the nerve and in the muscle the change from the state of rest to that of excitation is brought about by changes in the ionic concentration of the medium.[3] Lazareff termed this the ionic theory of stimulation, and on it developed a theory of nervous excitation of vision, hearing and taste. Recently he has on this basis formulated laws of the function of the central nervous system.[3] Surely, if ionization plays a rôle in nervous activity, the question as to the rôle of electricity is already answered. In a preparation of the sciatic nerve and gastrocnemius muscle of a frog, if the sciatic nerve be slightly pinched, the muscle contracts. Something must pass along the nerve from the point pinched to the muscle, and this has been shown to be an electric change.

> A spot in a state of excitation behaves as if electrically negative to a spot on the nerve at rest; that is, if the two points are connected to a galvanometer, a current flows through the instrument from the resting to the excited spot, as if the former corresponded to the copper of a Daniell battery and the latter to the zinc. We find that the electrical change set up at one point by a momentary stimulus lasts only for a short time at this point and passes along the nerve, making each point in turn electro-negative to the rest.[4]

The essential rôle of electricity must be admitted. And this must be true alike of the continuous neurofibrils of invertebrate forms and

3. Lazareff, P.: Science, New Ser., **59**:369 (April 25) 1924.
4. Bayliss: General Principles of Physiology, Ed. 3, London, 1920, p. 379.

of the synaptic nervous system of vertebrates. Finally, in the nervous system of vertebrates, the synapse has all the force and value of a Pupin inductance coil, and, like the latter, it both reenforces and facilitates transmission. Just as in the case of the Pupin inductance coil inserted in the telephone wire, the presence of the synapse in the nervous pathways of vertebrates results in an enormous economy of energy. Thus the interrupted synaptic nervous system of vertebrates enjoys an inestimable advantage over the nervous system of invertebrates dependent as the latter is on continuous and unbroken lines of transmission. We have here a repetition of the story of La Grange's weighted string.[5]

Other facts concerning nerve conduction may be briefly considered. Helmholtz long ago demonstrated that the rate of nerve conduction for the frog is 29 m. (about 95 ft.) per second. This is inconceivably slower than the rate estimated for electricity. It is probable that the process of ionic concentration requires time, and that considerable time is lost at the synapses. Apparently, time is consumed in the preparation of the synapse for transmission, i. e., in the "setting" of the synapse (Sherrington), which possibly consists in the formation of protoplasmic extensions, in the passage of ions, or in the establishment of induction. Many studies have been made as to the time lost in the passage through gray matter of various reflexes, and it would appear that the simpler the reflex, the shorter the reaction time; thus a simple spinal reflex in the frog reveals a loss of 0.008 second (Wundt) or 0.014 and 0.021 second (Buchanan), while the simplest reaction times measured in the psychologic laboratories vary between 0.1 and 0.2 second,[6] and the reaction time as measured by physicians between a "stimulus word" and a "reaction word" ranges from one to two seconds, sometimes longer.

In any event, enough is known to justify the correlation of nervous transmission with purely physical processes. That a nerve impulse is aroused by simply pinching a nerve, leaves no doubt as to the conversion of the impact into a transmission featured by ionization, in itself an electrical phenomenon. Finally, all living protoplasm is irritable, that is, reacts to physical and other impacts, as instanced in the sponge. Perhaps the great delay in transmission is due to the structure of living protoplasm itself.

The inference is that the phenomena of living protoplasm must be interpreted in the same terms as other physical phenomena. If electrical change, ionization, constitutes the underlying fact of transmission of impacts, such transmission is clearly expressive of the same

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5. Pupin, Michael: From Immigrant to Inventor, New York, Charles Scribner's Sons, 1923, p. 330.

6. Herrick: Introduction to Neurology, Ed. 3, Philadelphia, W. B. Saunders Company, 1922, p. 104.

force or forces observed in the inorganic world. That the forces of the latter are dependent on and inherent in the very structure of the space-time continuum itself, is clearly revealed by Einstein's interpretation.

INTERPRETATION OF ENERGY AND MATTER PHYSICAL AS WELL AS METAPHYSICAL

It may be thought that this interpretation of energy and matter is purely speculative and metaphysical. But the modern conception of the structure of the atom and of the constitution of the universe is not metaphysical. Certainly the deflection of a beam of light by the gravitational field of the sun is a fact definitely confirmed by a number of observers. At a meeting of the Royal Society, Nov. 6, 1919, Sir Joseph J. Thomson, the president, said:[7] "This is the most important result obtained in connection with the theory of gravitation since Newton's day. Einstein's reasoning is the result of one of the highest achievements of human thought."

Second, the displacement of the lines of the solar spectrum is another physical fact. If the position of the sodium line in a spectrum produced at the surface of the earth be compared with the position of the sodium line in the solar spectrum, we find that it has been displaced toward the red end of the spectrum, an effect clearly attributable to the action of the gravitational field of the sun. This fact which, as in the case of the deflections of the beam of light, was predicted by Einstein, does not embody a metaphysical conception; it is most assuredly physical.

Again, the perihelion of the planet mercury advances at the rate of 574 seconds of an arc in a century. According to mathematical calculation, this advance should be at the rate of 532 seconds. This leaves forty-two seconds to be accounted for. According to Einstein, this discrepancy is due to movement in a curved space. His specific contention is that in one revolution of the planet, the orbit will advance by a fraction of a revolution equal to three times the square of the ratio of the velocity of light. Einstein's calculations revealed forty-three seconds, leaving only one second as a possible error to be accounted for.[8]

Finally, Thomson, Rutherford, Milliken, Aston, Moseley, Bohr and other physicists have conclusively demonstrated the structure of matter. The atom has been resolved into its nucleus and revolving electrons.

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7. Harrow, Benjamin: From Newton to Einstein, New York, D. Van Nostrand Company, 1920, p. 41.

8. Jeans, James Hopwood: Encyclopaedia Britannica, New Volumes 32:261, 1922.

The nucleus, also termed proton, is electropositive, the electrons being electronegative. The structure of the atom of an element, other things being equal, depends on the number of its revolving electrons; thus the hydrogen atom possesses only one electron, while the other elements possess electrons in a progressively increasing number until we reach the heaviest and most complex of them all—uranium, which has ninety-two electrons revolving about its nucleus. It has even been possible to reduce the number of electrons of a given element and thus to change it into another. The very structure of the nucleus has been studied, and is revealed as being made up both of electropositive and electronegative factors, the electropositive predominating. The conclusion is inevitable that the atom with its nucleus and electrons is only a manifestation of electricity. Certainly nothing could be further removed than are such facts from the speculations of the Greeks or of our own metaphysicians of a century ago.

COMMENT

The interpretation of the structure of matter and of the universe opened up by Einstein must greatly influence our conceptions regarding living forms. The basic distinction between living and nonliving substance disappears. Our age-old conceptions regarding nonliving matter reveal themselves to be hopelessly at fault. The universe is in no part and at no time "dead" or "inert." Taken in its entirety or in its most minute subdivisions, it reveals itself in its ultimate analysis to be an expression of energy, a moving, pulsating, throbbing, "living" thing, a thing in which we are immersed and of which we form a part. It does not seem philosophical to regard those moving molecular aggregates which we term living forms, no matter how integrated and differentiated they may be, as things separate and distinct from the rest of the universe. Assuredly it does not seem necessary to evoke special agencies to account either for their existence or for the phenomena which they present. One of the generalizations held out to us by the conception of the universe outlined in this paper is that the universe is a whole, all parts of which are expressive of manifestations of energy, manifestations cognate and correlative and intrinsically identical. Of necessity, such a conception must include living matter.

TORULA MENINGITIS *

LOUIS L. SHAPIRO, M.D.
Instructor in Medicine, University and Bellevue Hospital Medical College
AND
JOSEPHINE B. NEAL, M.D.
Chief of Meningitis Division, Research Laboratory Department of Health, City of New York
NEW YORK

The classification of pathogenic yeasts has been the subject of much discussion among bacteriologists. As a result, the nomenclature has been greatly confused. In recent years, it has been shown that one of these forms, *Torula,* has a special predilection for the central nervous system and the lungs. The outstanding study of this organism and its rôle in producing infection has been made by Stoddard and Cutler.[1]

Earlier work was done by Türck [2] and von Hansemann [3] in Germany, and valuable contributions have also been made by Brewer and Wood,[4] Pierson,[5] Rusk,[6] Evans,[7] Freeman and Weidman,[8] Sheppe [9] and Bettin,[10] in this country. These workers have reported thirteen cases that may be considered due to the torula, although the cultural characteristics have not always been entirely identical. Sheppe, in a personal communication, reports another case of meningeal involvement.

This makes, with our own, a total of fifteen cases, of which thirteen involved the central nervous system, one the lung and one the muscles of the back and the vertebral column. Of these thirteen cases of involvement of the central nervous system, only five were diagnosed during life by the examination of the spinal fluid. In all cases except our own, the diagnosis was made only a comparatively short time before death.

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* Read at the Fiftieth Annual Meeting of the American Neurological Association, Philadelphia, June 6, 1924.

1. Stoddard and Cutler: Monograph of Rockefeller Institute for Medical Research, Jan. 31, 1916, No. 6.
2. Türck: Arch. Klin. Med. **90**:335, 1907.
3. Von Hansemann: Verhandl. d. deutsch. path. Gesselsch. **9**:21, 1906.
4. Brewer and Wood: Ann. Surg. **48**:889, 1908.
5. Pierson, P. H.: Torula in Man, J. A. M. A. **69**:2179 (Dec. 29) 1917.
6. Rusk: Univ. Calif. Pub. in Path., 1912, No. 2.
7. Evans: Calif. State J. Med. **20**:383, 1922.
8. Freeman, W., and Weidman, F. O.: Cystic Blastomycosis of the Cerebral Gray Matter Caused by Torula Histolytica, Stoddard and Cutler, Arch. Neurol. & Psychiat. **9**:589 (May) 1923.
9. Sheppe: Am. J. Med. Sc. **167**:91, 1924.
10. Bettin, Mona E.: Calif. & West. Med. **22**, No. 3 (March) 1924.

REPORT OF A CASE

History.—A boy, aged 16, born in New York, had spent most of his life in New York and New Jersey. He was 6 feet and 1 inch in height, and was well developed but slender. He was attending a private school. He had always been well, and there was nothing of moment in his past history until February, 1923, when he had complained of left frontal headache, weakness and sleepiness, which persisted for about one week. At this time, he did not appear to be

TABLE 1.—*Collected Cases of Torula Infection in Man (Authentic Cases)*

	No. of Cases	Sex	Age	Race	System Chiefly Involved	Result	When Diagnosis Was Made	Duration of Disease
1. Brewer and Wood..	1	M	20	Russian	Muscles and vertebral column	Recovered	Organisms recovered	5 months
2. Pierson.....	1	M	57	American, California	Central nervous system	Fatal	Post-mortem	6 weeks
3. Stoddard and Cutler	1	F	42	American, Florida	Central nervous system	Fatal	Post-mortem	14 weeks
4. Stoddard and Cutler	1	M	39	American, Massachusetts	Central nervous system	Fatal	Post-mortem	12 weeks
5. Rusk.....	1	M	57	German, California	Central nervous system	Fatal	Post-mortem	10 months? complicated by syphilis
6. Rusk.....	1	..	..	German, California	Central nervous system	Fatal	Post-mortem	1 month
7. Türck.....	1	F	43	German	Central nervous system	Fatal	Post-mortem	6 weeks
8. Von Hansemann...	1	M	18	German	Central nervous system	Fatal	Post-mortem	?
9. Evans.....	1	M	13	Mexican, California	Central nervous system	Fatal	Ante-mortem	5 weeks
10. Evans.....	1	F	20	Mexican, California	Central nervous system	Fatal	Ante-mortem	9 weeks
11. Freeman and Weidman	1	M	39	American, Pennsylvania	Central nervous system	Fatal	Ante-mortem	4 months
12. Sheppe.....	1	M	48	American, Virginia	Lungs	Fatal	Post-mortem	2 months
13. Sheppe (personal communication)	1	M	..	American	Central nervous system	Fatal	Ante-mortem	Few days ?
14. Bettin.....	1	F	40	American	Central nervous system	Fatal	Ante-mortem	5 weeks
15. Shapiro and Neal..	1	M	16	American, New York	Central nervous system	Fatal	Ante-mortem	7 months

suffering from any acute or chronic disease. His mentality was normal, and physical examination, especially of the nervous system, eyes, ears, nose and sinuses was negative.

Roentgen-ray examination of the head was negative. The temperature, pulse and respiration were normal. Blood pressure was: systolic, 120; diastolic, 68; hemoglobin (Dare) 95 per cent.; urine negative. Suspecting encephalitis, he was kept at home another week, at the end of which time he felt so well that he returned to school.

Course of Illness.—He was well until May 15, when, after eating a large quantity of chocolate and pie, he suddenly developed a similar left frontal headache with vomiting, weakness and dizziness. These symptoms persisting for several days, he returned to New York, and was again seen on May 19. Not responding to symptomatic treatment, he was sent to a sanatorium on May 23. Physical examination at this time was negative, except for a loss of 15 pounds (6.8 kg.) in weight since February. Mentally, he was normal until the evening of May 25, when he suddenly became delirious. This lasted but a few hours. Neurologic examination at this time showed exaggeration of both patellar reflexes only. On May 27, the patient complained for the first time of pain in the back of the head and neck, slight disturbance of vision and photophobia. From then until May 29, evidence rapidly developed that we were dealing with an acute infection of the central nervous system, particularly of the meninges. He showed definite rigidity of the neck. The eyes were painful on extreme movement to

A B

Fig. 1.—*A*, forty-eight hour culture of *Torula* on glucose veal agar plate; *B*, seven day culture.

the left, the pupils were equal in size and reacted normally to light and accommodation. The fundi showed beginning choked disk slightly more developed on the left. There was no definite evidence of cranial nerve involvement. The abdominal and cremasteric reflexes were diminished, especially the right. Both knee reflexes were markedly diminished. The bilateral Kernig sign was positive. Mentally, he was clear but somewhat irritable. The temperature was 101 F., pulse, 52; respiration, 20 and regular. The temperature and pulse previous to this time had always been normal. A cell count revealed: red blood cells, 5,640,000; hemoglobin, 89 per cent. (Dare); white blood cells, 14,400; polymorphonuclears, 80 per cent., and lymphocytes, 19 per cent.

Lumbar puncture was performed for the first time on May 29. The fluid was found to be under very high tension, and slightly hazy in appearance. There was no growth in the culture of the fluid the first eighteen hours, but distinct yeast colonies were apparent on the following morning, May 31, thereby establishing the diagnosis of a yeast infection of the central nervous system. Lumbar punctures were subsequently made daily until near the end, and the torula organism was recovered from the fluid by culture and observed in smears.

From May 29 until June 18, there was evidence of rapid progress of the disease, with symptoms of increasing intracranial pressure. The patient was frequently delirious and lost weight rapidly. He complained of photophobia and double vision. Rigidity of the neck persisted. The pupils were moderately dilated and were sluggish to light. Distinct weakness of the right and left external recti developed. The fundi showed increasing papilledema, being from 2 to 3 diopters on June 2, and 4 or 5 diopters on June 7. The abdominal and cremasteric reflexes were not elicited. The deep reflexes were generally diminished. A bilateral Kernig sign was present, but no Babinski sign. The temperature was irregular, varying from 98.6 to 103.8 F. The pulse was always slow in comparison with the temperature, ranging from 60 to 100, and averaging about 75. Respiration was normal. On the morning of June 8, the patient went into a deep stupor. Toward evening, he gradually roused after another lumbar puncture and tube feeding, and was rational again the next morning. Tube feeding was necessary from this time on. This was done every six hours, and continued until about the middle of August. In this way, the caloric intake for twenty-four hours was increased from less than 800 to more than 5,000. Up to this stage, one lumbar puncture daily was sufficient to relieve intracranial pressure. Now, however, a puncture would afford relief for only about ten hours, when the patient would again become restless, irrational, noisy and cry out as if in great pain. Punctures therefore were made twice a day until June 19, when one puncture again gave sufficient relief.

Tube feeding was discontinued about the middle of August, from which time he took nourishment well, averaging about 5,000 calories in twenty-four hours. He gained considerably in weight and appeared to be well nourished. He was quiet and seemed comfortable most of the time. Mentally, he was for the most part lethargic, confused and disoriented. Frequently, there were periods lasting several days or weeks when at times he seemed quite rational, would talk coherently when spoken to, would recognize people, and appeared to be interested in what was going on about him. He rarely complained of headache and vomited only three times. Weakness of the right and left external recti persisted throughout. The fundi improved considerably, showing an elevation of 1 diopter in the right disk and 1.5 diopters in the left. The margins appeared fairly distinct, and the veins were less tortuous and full. No further cranial nerve involvement appeared. The abdominal and other superficial and deep reflexes remained absent throughout. Kernig's sign was always present. No gross sensory or motor changes were obtained. The temperature was always irregular, showing a morning fall and evening rise. From the middle of June to early in July, it varied from 101 to 103.6 F., occasionally reaching 104 and once 105 degrees. During the remainder of July, it varied from 100 to 102 F., except for a brief interval. During August and September, it varied most of the time between 99 and 101 F., occasionally reaching 102 degrees during September.

The pulse gradually increased in rate, varying during the last half of June between 100 and 120. During July, it ranged between 110 and 130. During August and September, it varied between 130 and 150, averaging about 140. The respirations were always normal and regular. During this entire period, the lungs were clear, and the heart was normal except for the tachycardia. The blood pressure varied from time to time between 90 and 144 systolic.

During the last week in September, scattered, moist râles were detected at both bases posteriorly. On October 1, the patient again became stuporous. The lungs showed impaired resonance, with small areas of distant, high pitched

breathing. Pulmonary edema developed on October 8, and the patient died on the evening of October 11 of respiratory failure.

Peculiar pressure sores developed over both hip joints, the inner side of both knee joints and both heels during the last month of the disease. They began as large violet blue blebs, rapidly breaking down, becoming excavated, but with firm indurated margins. Cultures taken from these areas were negative for torula organisms.

Treatment.—As the smear from the first spinal fluid withdrawn showed forms suggesting gram-negative cocci, antimeningococcic serum in 20 c.c. doses

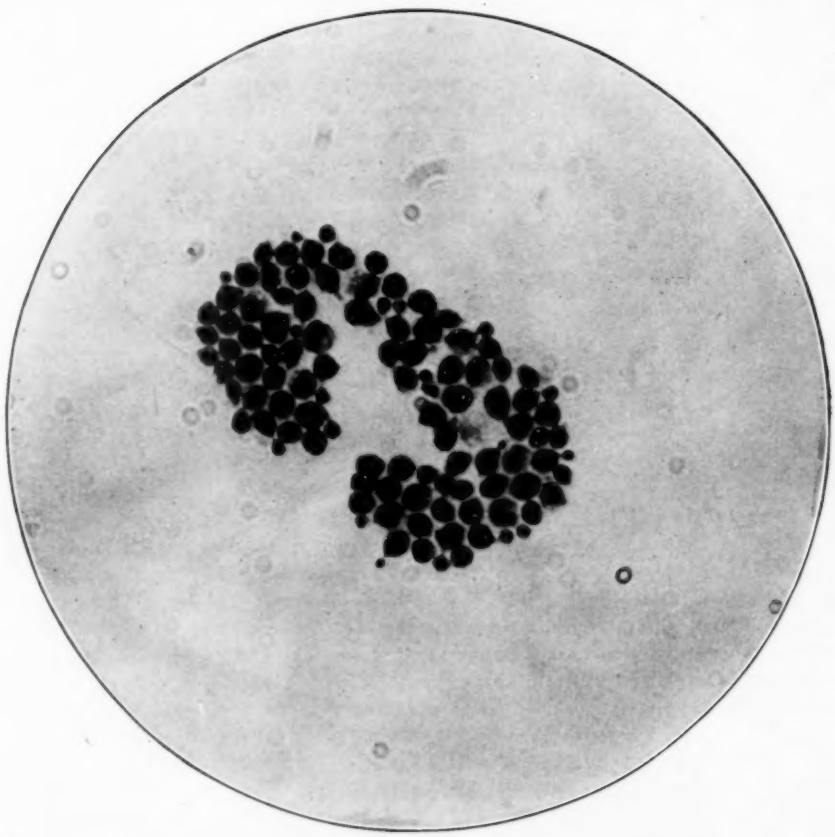

Fig. 2.—Smear of fifteen day culture of *Torula;* budding forms.

was given intraspinally the following four days. When the diagnosis of yeast infection was definitely established, it was decided to give iodids because of their action on other yeast infections, especially the oidiomyces. Sodium iodid, 15½ grains (1gm.) was therefore given intraspinally every other day, beginning June 3, until July 19. One hundred grains (6.5 gm.) of sodium iodid were also given daily internally, beginning May 31, and increased to 400 grains (26 gm.) daily from August 1 to the end of September. The iodids were given again intraspinally every other day during August. The patient tolerated the

iodids very well, and at no time showed symptoms of iodism. Because of the favorable reports from the use of monilia vaccine in tropical sprue, a vaccine was made from the torula organisms and given twice weekly, beginning June 6 with 500,000, increasing 500,000 with each dose. This was continued throughout the entire course of the disease. Beginning August 25, vaccine prepared by the Dreyer method was used. The patient never showed any local or general reaction.

On July 21, 24 and 26, a colloidal silver preparation was given intraspinally. Each administration was followed in a few hours by a thermic reaction, the temperature rising to 103 F. and gradually declining to 99 F. in forty-eight hours. This was tried on account of a great increase in the number of colonies in cultures. After the first injection, there was also a severe general reaction. As there was no evidence of improvement in the patient's condition or of effect on the growth of the organisms, the colloidal silver preparation was discontinued after the third dose.

Serum obtained from rabbits immunized by intravenous injection of the torula was given intraspinally on September 5, 8 and 13, the first dose being 5 c.c., the second 3 c.c., and the third 5 c.c. The third dose was followed by a severe general reaction, with a temperature of 104 F., pulse 170 and respiration 30, so that it seemed inadvisable to continue giving the serum.

Arsenic was given internally in the form of liquor potassii arsenitis (Fowlers' solution), 15 minims (1 c.c.) daily during the month of August.

A high caloric diet averaging about 5,000 calories in twenty-four hours, well proportioned in proteins, carbohydrates, fats, fruit juices and salts, was given and well tolerated throughout the entire course of the disease. As mentioned previously, tube feeding was resorted to for a period of about two months.

Clinical Pathology—The Wassermann test was negative on the blood and the spinal fluid. Blood cultures taken June 7 and 29, August 21, September 23 and October 5 were all negative for *Torula*. Complete blood counts were made every ten days throughout the course of the disease. The average count for June was: hemoglobin, 80 per cent. (Dare); red blood cells, 4,840,000; differential count normal; color index, 0.83; white blood cells, 12,400; polymorphonuclear neutrophils, 83 per cent.; lymphocytes, 13 per cent.; large mononuclears, 4 per cent.; eosinophils, none.

On June 8, when the patient was in deep stupor, the white blood cells were 28,000 with 94 per cent. polymorphonuclears. The average counts for July, August and September were about the same. On October 10, the examination showed for the first time qualitative changes indicative of anemia.

The blood chemistry on June 11 was within normal ranges. The findings were: nonprotein nitrogen, 34 mg. per 100 c.c. of blood; urea nitrogen, 15 mg. per 100 c.c. of blood; uric acid, 2.5 mg. per 100 c.c. of blood; blood creatin, 1.5 mg. per 100 c.c. of blood; blood sugar, 100 mg. per 100 c.c. of blood; whole blood chlorid 363 mg. per 100 c.c. of blood; alkali reserve, 70 per cent.

On August 16, an agglutination was made with the torula and the patient's serum, undiluted, and in a dilution of 1:10, 1:50, 1:100 and 1:500. There was no agglutination, although the patient had been receiving vaccine for more than two months. Agglutination in the same dilutions were made with horse and sheep serum. It was hoped that one of these animals would show some natural immunity to the torula, but none was found.

Throat cultures and cultures of the sputum were negative for torula organisms.

Urine analyses were made practically daily throughout the disease. The average analysis was: specific gravity, 1,023; albumin, trace, with few medium to large finely and coarsely granular and hyaline casts; sugar, acetone and diacetic acid, none.

Cultures of the urine from time to time were negative for *Torula* organisms. The patient had a urethral discharge during September. Cultures from this were negative for *Torula* organisms and gonococci.

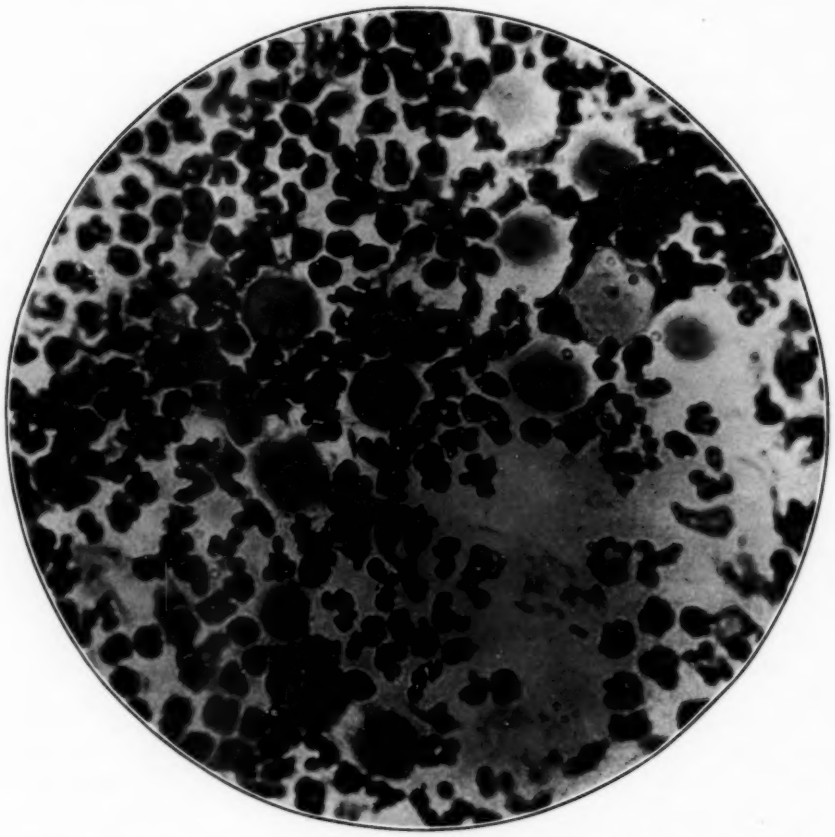

Fig. 3.—Smear of ninth spinal fluid, showing forms of various sizes. Rings of cytoplasm vary in width. Nuclear material is granular in some forms.

Spinal Fluid Examinations.—The first lumbar puncture was performed on May 29, 1923. The fluid was under greatly increased pressure and was slightly hazy and showed 300 cells per cubic millimeter. Of course, many of these were yeast cells. The other cells were about 90 per cent. mononuclears. Smears were stained both by the Ziehl-Neelson stain and by the Gram method. No tubercle bacilli were found in the Ziehl-Neelson stain, but there were many large heavily stained cells which were later identified as yeast cells. In the smear stained by the Gram method, there were seen degenerated cells, with a suggestion of intracellular gram-negative cocci. There was a moderate increase

(++) in albumin and globulin in the fluid, and the sugar content as shown by the reduction of Fehling's solution was markedly diminished (+).

The second lumbar puncture, made May 30, revealed 45 c.c. of hazy fluid, with practically the same findings as in the first, except that the percentage of polymorphonuclear cells was considerably higher.

On May 31, there was a heavy growth of yeast on the forty hour culture from the first fluid and a light growth on the twenty-four hour culture from the second fluid. The smears and cultures were submitted to Dr. Anna W. Williams, assistant director of the research laboratory.

Daily lumbar punctures were performed until June 8. At each puncture, from 30 to 60 c.c. of fluid were withdrawn. The chemistry of the fluid remained about the same, but the type of cells was apparently modified somewhat by the administration of serum, as fluids withdrawn after serum was given usually showed a high percentage (80 to 90 per cent.) of polymorphonuclears. A Wassermann test on the second fluid was negative. The p_H of a number of fluids was ascertained. It ranged from 7.5 to 7.8, which is within the normal limits for spinal fluid and indicates that the torula was not rendering the fluid acid. In the first four fluids, the yeast cells were large and stained heavily. The structure was not well shown. They stained best by the Ziehl-Neelson method. With the Gram stain, they were amphophilic and less easily distinguishable from the cells of the spinal fluid. After the fourth puncture, the structure of the torula was better shown. The organisms varied greatly in size and appearance. In some, the margin was sharply defined; in others, the outline was indistinct and fuzzy. In some, the greater part of the cell was made up of a densely staining nuclear-like material, surrounded by cytoplasm of varying widths. In others, this densely staining nuclear substance was relatively small and surrounded by a wide clear cytoplasm. At times, the center of the cell was apparently broken up into granules, which often took a reddish stain. Budding forms were seen on practically all types of cells. Small forms with practically no cytoplasm might have been mistaken for small lymphocytes if buds had not been seen. The cytoplasm was apparently of a gelatinous nature, as shown by the fuzzy halo. This gelatinous character may also account for the fact that the yeast cells, especially those with a small nucleus and a wide cytoplasm, were often almost completely surrounded by lymphocytes. The gelatinous character of the cytoplasm or capsule has also been noted by Sheppe, who observed that in contaminated cultures the yeast cells were often surrounded by other organisms. In all of these early cultures, there was a luxuriant growth in from twenty-four to thirty-six hours.

From June 8 to June 18 inclusive, two punctures were made daily for the relief of pressure. From 40 to 60 c.c. of fluid were usually withdrawn at each puncture. These fluids yielded practically the same findings as before. On June 15, the fluids became somewhat clearer, with a corresponding decrease in tissue cells—although they remained greatly increased above the normal. The relative proportion of mononuclears and polymorphonuclears often changed for no apparent reason. The number of yeast cells varied from time to time. On June 16, the culture began to be less luxuriant, and from that time the number of colonies gradually decreased in number and did not appear until forty-eight hours had elapsed. Beginning with June 19, daily punctures were made again. The character of the fluid showed little change, but the colonies continued to diminish in number, and on June 21 did not appear until after seventy-two hours had elapsed. The number of colonies was small, even on plates inoculated with centrifuged sediment. For some time, plates had been made both with the

centrifuged and uncentrifuged fluid to estimate as well as possible the number of yeast cells. It was noted that there was little relation between the number of colonies and the number of yeast cells seen in the smear. Often when only two or three yeast cells could be found in the smear, there was an abundant growth, and we therefore concluded that it was impossible to differentiate the small form of the torula from the lymphocyte. This decrease in the number of colonies and their rate of growth continued until July 16, when the cultures

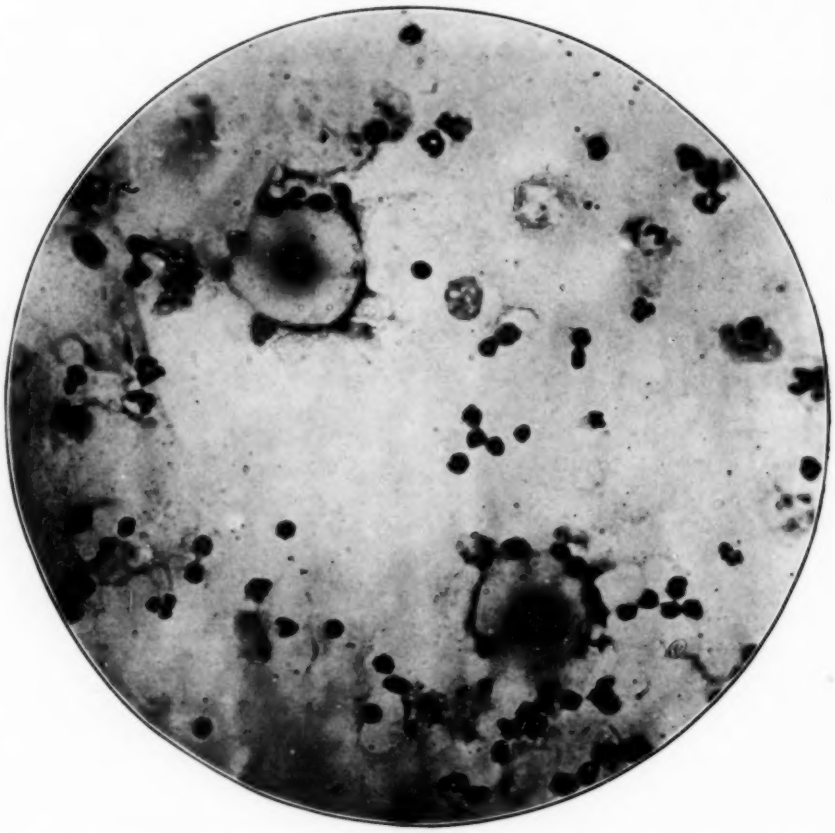

Fig. 4.—Smear of twenty-fourth spinal fluid showing *Torula* surrounded by lymphocytes.

again began to show in forty-eight hours and the number of colonies began to increase rapidly. On August 1, the colonies grew in twenty-four hours. From this time, the colonies varied in rate of growth and in number, but they were on the whole far less numerous than during the last two weeks of July. The cultures continued positive until September 26, although the number of colonies was small. The cultures on that date were negative, as were also the succeeding five cultures. The character of the fluid remained much the same until August 22, when the fluid became slightly blood-tinged. On August 26, the fluid was yellowish, with no free blood present. This condition continued for some

days, when the fluid again became slightly blood-tinged, and thereafter was either blood-tinged or yellowish until the end. The amount of blood present apparently was not sufficient to affect greatly the protein content, since the quantity of albumin and globulin gradually decreased from a moderate (++) to a slight (+) amount by the end of August. The amount of sugar gradually increased and was apparently normal by the middle of August.

On September 6, after an intraspinal injection of rabbit serum, the quantity of albumin and globulin increased, and the percentage of cells changed from 70 to 80 per cent. mononuclears and to 70 per cent. polymorphonuclears. On September 14, following the reaction to the rabbit serum, the quantity of sugar became suddenly greatly diminished, but subsequently increased to normal and remained so until the last fluid withdrawn on October 8, when it again fell off slightly.

From time to time, a web appeared in the fluid. At times it was fine, resembling the web in the fluid from a case of tuberculous meningitis. But at times it was dense and gelatinous, with many branches extending from a central filament to the sides of the tubes.

In all, 133 lumbar punctures were performed. Except at the last two punctures, when a higher level was used, the punctures were made in three interspaces—between the fourth and fifth, third and fourth and second and third lumbar vertebrae, and the back remained in excellent condition. Except for some staining of the skin from the iodin, there was little indication that it had been submitted to such a long continued ordeal.

The cultures grew readily on ordinary agar either with or without the addition of blood. The p_H of the mediums did not seem to affect the growth. Mediums of various p_H values were tried ranging from 5.8 to 8.6, with no apparent effect on the growth. We used 2 per cent. glucose veal agar, p_H 7.6. The cultures grew equaly well at room and at incubator temperatures. When the colonies first appeared, they were pin point in size and increased rapidly to perhaps a centimeter in diameter. The discrete colonies were uniformly rounded, with a sharply defined margin and a high center. They appeared moist and glistening, and did not adhere tightly to the medium. In consistency they were soft and not stringy. On slants, they coalesced, forming a thick moist, creamy growth. In color, they were a very pale creamy yellow at first, becoming darker yellow as they grew older. The tops of the older colonies were darker than the rest. They grow less well in beef extract broth, p_H 7.5, than on solid mediums. They clouded the broth slightly, and formed a heavy, creamy sediment in the bottom of the tube. There was no pellicle formation. The cultures did not grow anaerobically. No mycelial formation was noted. The action on sugars was studied by surface and stab cultures in veal agar p_H 7.4 containing 1 per cent. of the various sugars and 1 c.c. Andrade. There was no formation of either gas or acid at the end of more than thirty days in the following sugars: lactose, levulose, mannite, glucose, saccharose, inulin, dextrose, starch and glycerin.

The smears of the cultures were gram-positive in the earlier generations, but smears made from cultures several months old showed many gram-negative forms. In the earlier cultures, the organisms had a deeply staining center, with a narrow ring of less deeply staining cytoplasm. While they varied greatly in size, no large forms, such as were found in the smears of the spinal fluids, were ever seen. In the older cultures, the central material was broken up into granules. Many budding forms were seen in the cultures. There was no evidence of endospores.

The absence of endospores, the reproduction by budding, the absence of mycelium and the failure to form gas in sugars identify the yeast as a torula.

Park and Williams [11] give the following tentative classification of the yeast and the so-called intermediate groups.

Somewhat rough studies were made in an effort to find some substance that might have an inhibiting effect on the growth of the torula, with the following results: sodium iodid, 1:500, no inhibition; sodium salicylate, 1:500, no inhibition; quinin, saturated solution—no inhibition; a colloidal silver preparation 1:600; 1:1,000; 1:6,000; slight inhibition; magnesium sulphate, 1:500, no inhibition; rochelle salts (tartrates) 1:500, no inhibition; tricresol, 1:1,000 some inhibition; acriflavin, 1:1,000; 1:5,000 complete inhibition. 1:10,000 well marked inhibition. Acriflavin was the only substance of the series which had any definite effect on the growth of the torula, but it was not used in treatment.

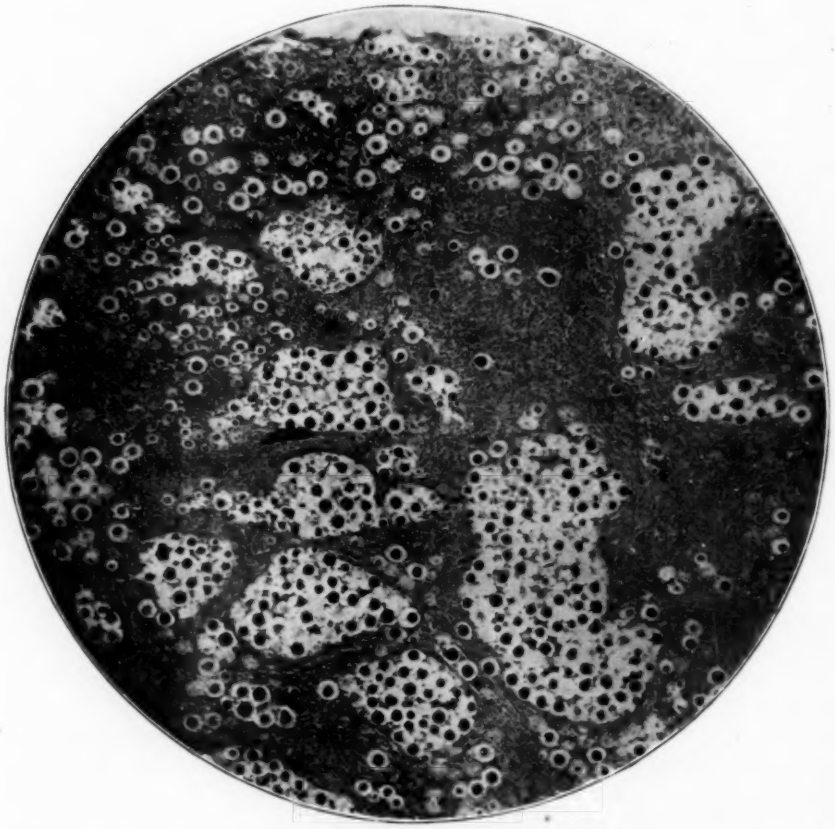

Fig. 5.—Low power magnification of section of subcutaneous mass, Guinea-Pig 644, showing *Torula* arranged in groups. Eosin-methylene blue stain.

11. Park and Williams: Pathogenic Microorganisms, Philadelphia, Lea & Febiger, 1920.

ANIMAL INOCULATION

On June 8, 1923, eight young animals, four rabbits (about 1,500 gm.) and four guinea-pigs (about 200 gm.) were inoculated with a heavy suspension of a forty-eight hour culture of the torula as follows: Rabbit 86, 0.25 c.c. intracranially. Rabbit 36, ¼ c.c. intracranially. Rabbit 621, 5 c.c. intraperitoneally. Rabbit 829, 3 c.c. intraperitoneally. Guinea-pig 657, 0.25 c.c. intracranially. Guinea-pig 655, 0.25 c.c. intracranially. Guinea-pig 647, 3 c.c. intraperitoneally. Guinea-pig 653, 2 c.c. intraperitoneally.

Rabbit 86 died, July 17, 1923, and no torula were found by culture in any of the organs.

Guinea-pig 657 died, June 17, and necropsy was not performed. Guinea-pig 653 died, June 22, and showed no torula in cultures.

Rabbit 829 seemed sick about August 27. This animal had been inoculated intraperitoneally about eleven weeks previously. There was a skin lesion on the back over the lower part of the spine, with superficial ulceration and crust formation. He had general muscular tremors, was ataxic in his movements and seemed to be blind. He was chloroformed and necropsy performed on August 30 by Mary Nevin. Just before death a lumbar puncture was performed,

TABLE 2.—*Park and Williams' Classification of Yeasts*

	Genus	Endospores	Budding	Mycelium	Gas Formation
Blastomycetes	Saccharomycetes	+	+	—	+
Fungi Imperfecti	Torula	—	+	—	—
	Monilia	—	+	±	+
	Oidia	—	+	+	—
	Coccidioides	+	—	+	—

and about 1.5 c.c. of spinal fluid withdrawn, which was hazy, showed a slight increase in mononuclear cells and in albumin and globulin and a normal sugar content. The smear showed a few torula and other organisms. The torula was finally recovered in pure culture from the fluid and also from the skin lesion, brain, liver, spleen, lungs and kidney. The organs in the gross showed no definite lesion. The sections showed no large forms of torula, but a study of the sections showed small forms similar to those in the culture scattered through the organs, especially in the periphery of the organs and in the lymph spaces of the brain.

Rabbit 36 died on October 16. It had been inoculated intracranially over four months previously. It had not appeared sick previously. It had apparently grown normally since the inoculation and was well nourished. On necropsy, the brain apparently was normal. The liver was pale, mottled and brittle. The spleen was enlarged and very dark. The kidneys were normal. The lungs showed edema, with some hemorrhagic spots and congestion. The torula was obtained in pure culture from the brain, liver, spleen, lungs and heart. The plates were inoculated by smearing sections of the organs over them. The colonies were numerous, and there was no contamination. As in Rabbitt 829, no large forms of torula were found in the sections of the brain, but small forms were found as in Rabbit 829. The other organs were not sectioned. It will be observed that in Rabbit 829 inoculated intraperitoneally, the torula had invaded not only the abdominal viscera and lungs, but also the spinal fluid and

brain. In Rabbit 36, inoculated intracranially, the torula had invaded not only the brain, but also the abdominal and thoracic viscera. It is also interesting to note that Rabbit 36 lived more than four months, and in spite of the widespread distribution of the torula, grew normally in size, was exceptionally well nourished and showed no evidence of infection up to the time of his death. The fourth rabbit apparently escaped or overcame the infection. He died, Jan. 18, 1924, over seven months after inoculation, and no torula were recovered by culture from any of the organs.

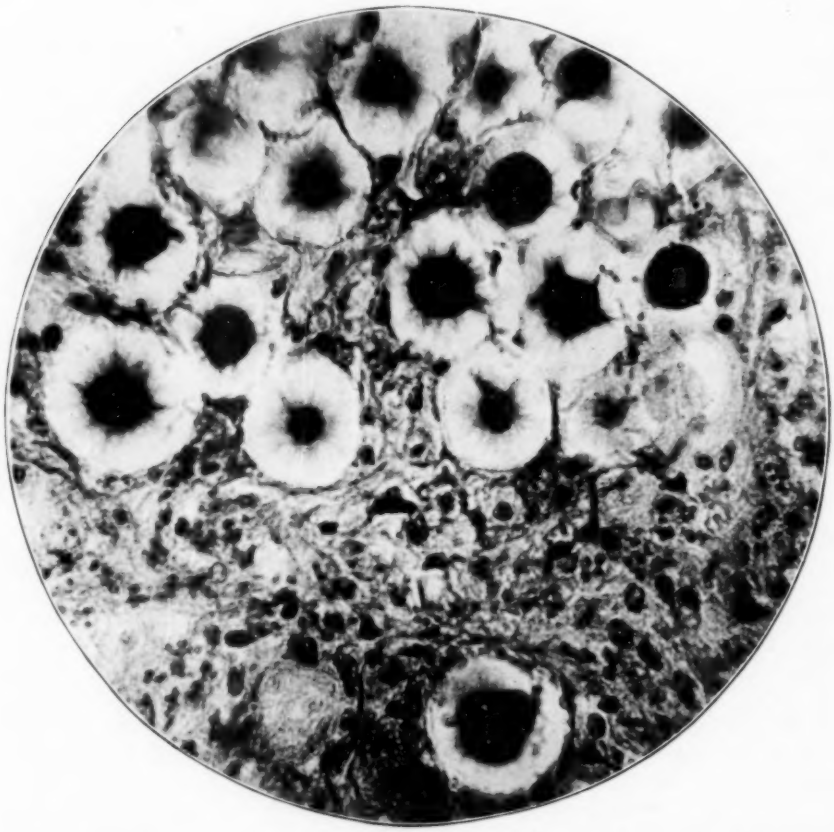

Fig. 6.—High power magnification of section in Figure 5. There are numerous *Torula*—a few budding forms.

The two guinea-pigs that survived grew rapidly and seemed perfectly well. They were kept under observation, however, and on November 3, more than five and one-half months after inoculation, it was noticed that Guinea-pig 655, inoculated intracranially, showed spasmodic contraction of groups of muscles, and held the head turned markedly to one side. He was weak in his movements and was unable to stand normally. He was panting for breath. He was chloroformed on that day and necropsy was performed. The brain was edematous and soft, and the fluid oozed out of it. The right lung was small and shrunken;

the left was swollen. The spleen was small and mottled. The stomach was empty and distended with gas. The other organs seemed normal. The torula was obtained in pure culture from the brain, lungs, liver, spleen, kidney and heart. The plates were inoculated by smearing with pieces of the organs. The colonies were numerous, and the culture showed practically no contamination.

In the sections from Guinea-pig 655, many large forms of the yeast were found, especially in the lymph spaces of the brain and in the meninges, and also in the lung (Figs. 7 and 8). It will be noted that budding forms are also seen.

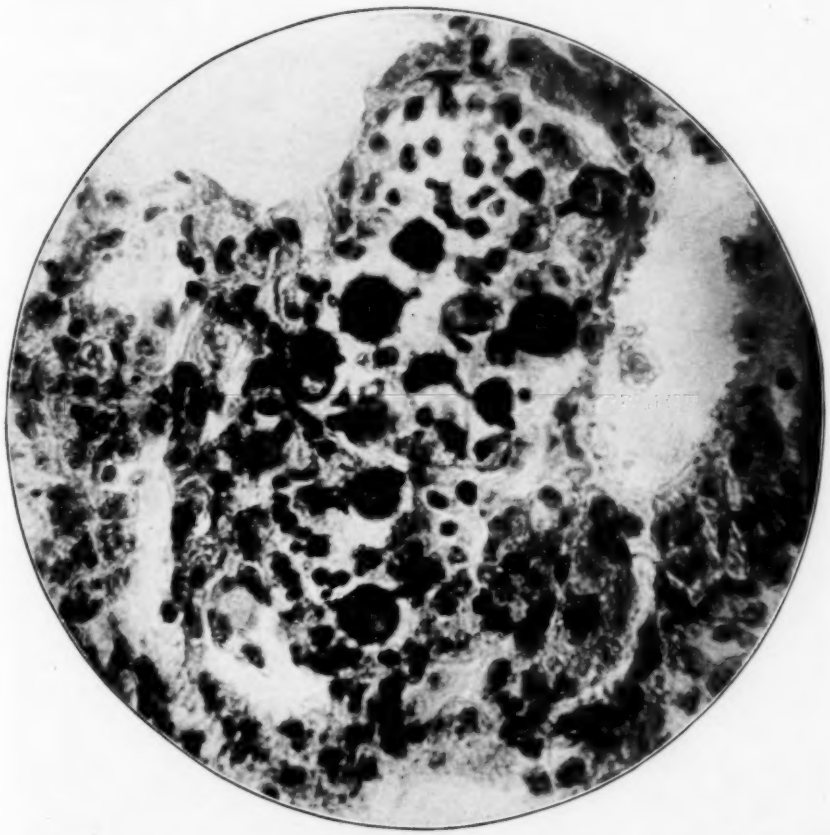

Fig. 7.—High power magnification of section of lung of Guinea-Pig 655. There are numerous *Torula*; a few budding forms.

Guinea-pig 644 seemed normal until late in December, when a hard mass was observed in the left abdominal wall. Soon after that, small ulcerative lesions were observed on the right lower eyelid and on the right cheek. He was chloroformed and necropsy was performed on Jan. 3, 1924, nearly seven months after the intraperitoneal inoculation. The brain and other organs seemed normal, and no torula could be cultivated from them. The torula was cultivated from the mass in the abdominal wall, and from the two skin lesions. The plates were inoculated as in the previous cases. The colonies were

numerous and the culture practically pure. All these cultures were tested with the sugars (mannite, glucose, glycerin, starch, saccharose, dextrin, inulin, lactose and levulose) and gave no fermentation with any of them. The mass in the abdominal wall was located in the subcutaneous tissue between the skin and the abdominal muscles. It was about 2 inches in length and 1 inch in diameter. It was irregularly spindle-shaped and about the consistency of

Fig. 8.—Low power magnification of section of brain of Guinea-Pig 655, showing *Torula* in lymph spaces around blood vessels and in meninges. Eosin-methylene blue stain.

paraffin. In the sections of this mass many large forms of torula were present. The arrangement in masses is shown in Figures 5 and 6. It will be noted that there is practically no cellular reaction accompanying the invasion with the torula. The torula was much better shown by the eosin-methylene blue stain than by the hematoxylin-eosin.

CONCLUSIONS

In comparing our case with those reported by others, it may be pointed out that we were fortunate in having the patient under our observation during the entire course of the illness. The diagnosis was made within the first two weeks of the acute illness and within five days of the onset of symptoms involving the central nervous system. It is possible that the real onset of the infection was in February, with a period of quiescence lasting until May. He lost 15 pounds in weight during this time, although otherwise apparently well.

Various methods of treatment were tried, apparently with no effect. Arsphenamin had been used by Stoddard and Cutler without success in one of their cases, and was not tried in our case. Decompression was considered, but the repeated lumbar punctures seemed to afford sufficient relief of pressure.

In spite of the fact that the boy was acutely ill for five months, with a constant fever, it was possible to keep him exceptionally well nourished until the very end, and there were very few focal symptoms, although the disease must have been constantly progressing. These same points were observed in the animals inoculated as has already been mentioned.

From a study of the patient and the animals inoculated, infection with this torula seems to be unsually slow and insidious in its course, and inevitable in its outcome.

DISCUSSION

Dr. Harvey Cushing, Boston: The first two cases of torula infection of the meninges, so far as I know, were reported in a monograph (No. 6) of the Rockefeller Institute Series in 1916, by Drs. E. C. Cutler and James Stoddard. The cases were those of patients in my clinic at the Brigham Hospital who were thought to have brain tumors. Both of them, as I recall it, had definite symptoms of intracranial pressure without any localizing features, and were operated on without avail. The diagnosis was made only at necropsy.

We have since had a third case in the Brigham Hospital, a patient on the medical wards, where, in spite of pressure symptoms, they are less conservative about lumbar punctures. Unfortunately, the clinical clerk who examined the fluid failed to recognize that the cells were torulas, and they were recorded as lymphocytes, which the fluid held in excessive number. The condition certainly should have been diagnosed before necropsy, although I do not know that this would have had any influence on the progress of the malady, which appears to be a fatal one.

I regret that I must tell this, but it gives me an additional opportunity to congratulate Dr. Tilney on having made the correct diagnosis in his case. At the same time, I wish to emphasize the need of great caution when cerebrospinal fluid, which shows no growth on culture to ordinary mediums, is said to contain an unusually large number of cells which are regarded as mononuclear lymphocytes.

Dr. Charles A. McKendree, New York: In conjunction with the paper presented by Drs. Shapiro and Neal, we thought it might be of interest to

demonstrate in a brief, preliminary and gross way our observations in a brain which shows leptomeningitis and invasion of the cortex by the yeast fungus.

Our report must necessarily be brief, because we have had the brain for two and a half weeks. The duration of the disease was nine months.

The patient, a woman, aged 50, began with gradual and progressive involvement of the external and internal ocular muscles in September, 1923. She came to the Neurological Institute four months later complaining of drowsiness, headache, vomiting and inability to move the muscles of her eyes. An extensive study was made of the patient at that time, and, not to burden you with any negative findings, I shall tell you only the positive observations. She had a complete external and internal ophthalmoplegia, 10 cells in the spinal fluid, and yeast cells.

At the time, most of us felt that we were dealing with an encephalitis of an inflammatory, progressive type, involving chiefly the nuclei of the cranial nerves. Dr. Tilney, however, reminded us that the finding of yeast cells in the spinal fluid might not be what we had a tendency to think — a contamination — and he urged further study from this angle. Unfortunately, because of the patient's condition, we could not make further spinal fluid findings. The patient left the hospital, since which time I have followed the case, and she died in May, 1924.

We have not been able to make any extensive studies, but Dr. Leon Cornwall, pathologist of the City Hospital, has made some frozen sections and will demonstrate the findings to date.

Dr. Leon Cornwall, New York: This brain was received by us only four days ago. Therefore it was possible for us to make only a few frozen sections. Two small segments of brain tissue were taken for examination, one from the right superior temporal convolution and the other from the left midfrontal region.

From this limited study, we feel justified in saying that this is a case of meningo-encephalitis due to yeast, the latter having been demonstrated in the sections. We hope, after further study, to be privileged to demonstrate to you further details of the pathology.

Dr. Josephine Neal, New York: What cultural work was done in Dr. McKendree's case; was the further study of yeast carried out?

Dr. McKendree: No cultural method was used in the case. The yeast was reported two days after the fluid was taken, and because of circumstances we could not resort to another puncture.

AN UNUSUAL CORTICAL CHANGE IN CARBON MONOXID POISONING*

GEORGE WILSON, M.D. AND N. W. WINKELMAN, M.D.
PHILADELPHIA

Among the unusual pathologic findings which occur secondary to carbon monoxid poisoning are changes in the peripheral nerves [1] and in the cortex.[2] Involvement of the globus pallidus is probably the commonest of all the pathologic changes that result from carbon monoxid poisoning. A careful search through the literature has revealed but one case similar to that which we report below; this was by Stewart.[2]

REPORT OF A CASE

History.—A woman, aged 25, white, married, was admitted to the University Hospital on Aug. 18, 1923, to the service of Dr. Alfred Stengel, to whom we are indebted for the privilege of reporting this case. She was brought in unconscious in a patrol wagon, and the police said that she had been in a room with the gas turned on for one hour and fifty-five minutes. She had cyanosis and was almost pulseless. Artificial respiration was necessary for a time, and the pulse and respiration improved. She had generalized muscular twitchings, and the pupils were dilated.

Examination.—One day after admission, respiration was abdominal, and the patient could not be aroused. No cranial nerve palsies were present. The mouth was tightly closed, and could be opened only with considerable force. The face was flushed. Four days after the exposure to gas, the extremities were flaccid. The biceps, triceps and patellar reflexes were present and about normal. Babinski's and Kernig's signs and ankle clonus were absent.

Laboratory Examinations.—The urine showed a trace of albumin and many white blood cells. There were 4,420,000 red blood cells, 15,100 white blood cells and 81 per cent. hemoglobin. Differential count showed no deviation from normal.

Clinical Course and Outcome.—Ten days after admission she did not respond to questions. The mouth was tightly closed, and all the extremities were flaccid. The deep reflexes were present and about normal, although the biceps and triceps were slightly exaggerated. Double foot and wrist drop were present. Plantar stimulation produced prompt flexion on both sides. She cried during the examination, but it was a question whether this was due to squeezing the nerves or to involuntary emotionalism. At this time, a diagnosis of multiple neuritis and cerebral involvement secondary to carbon monoxid poisoning was made.

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*From the Neurological Department of the School of Medicine of the University of Pennsylvania and the Laboratory of Neuropathology of the Philadelphia General Hospital.

1. Wilson, George, and Winkelman, N. W.: Multiple Neuritis Following Carbon Monoxid Poisoning, J. A. M. A. **82**:1407-1410 (May 3) 1924.

2. Stewart, R. M.: J. Neurol. & Psychopath. **1**:105, 1921.

Seventeen days after the onset, the patient was in a dying condition; the left pupil was dilated and the right pupil contracted. Attempts to open the mouth were followed by reflex closure of the jaws. A tremor of the jaw had disappeared. She died Sept. 4, 1923.

Pathologic Study.—Gross examination after fixation showed nothing but a fine gray line running parallel to the cortex in the middle of the gray matter, present throughout the entire brain and broken here and there by bridges of normal tissue, variable in width.

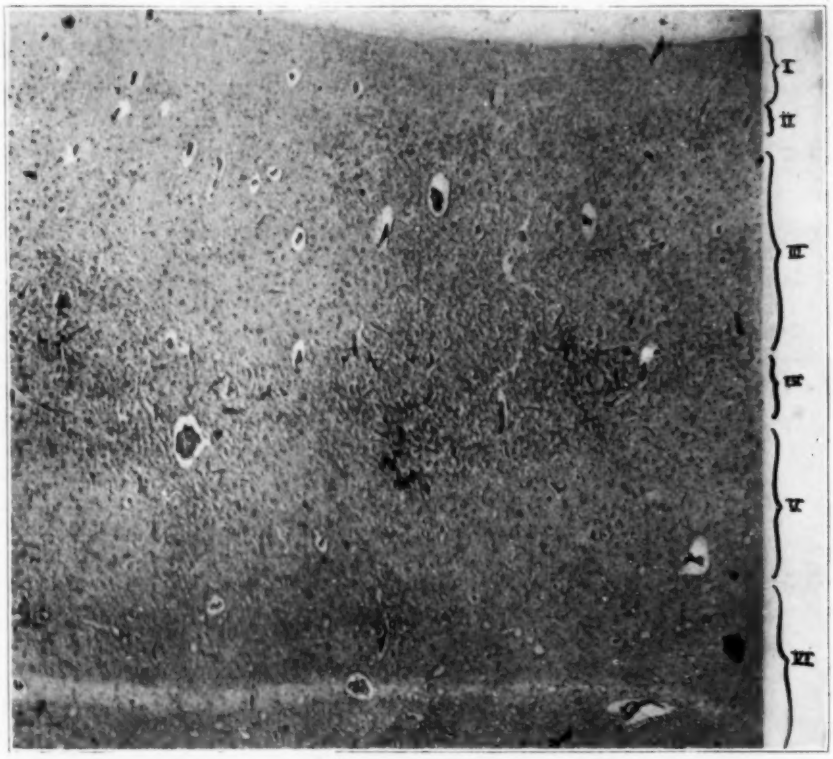

Fig. 1.—Low power magnification of characteristic area of cortex showing involvement of lower part of third and fourth layers. Toluidin blue stain

Microscopic sections from all parts of the cortex, both cerebrum and cerebellum, as well as the basal ganglia, were studied with the following staining methods: toluidin blue, hematoxylin-eosin, Bielschowsky, phosphotungstic-acid-hematoxylin, Herxheimer's scharlach R and Weigert's myelin sheath stain. Over the entire cortex, the pia-arachnoid was hyperplastic and in places infiltrated with phagocytic and mesothelial cells. The vessels were congested. The gray line which was just visible grossly proved to be a zone confined to the lower part of the third and the entire fourth cortical layers (Brodmann's classification) (Fig. 1). This area appeared to be much more vascular than the rest of the cortex (Fig. 2), and on higher magnification new capillary formation could be made out (Figs. 3 and 4). This was not an area of softening in the usual sense, because a comparatively normal cyto-architecture could be made out

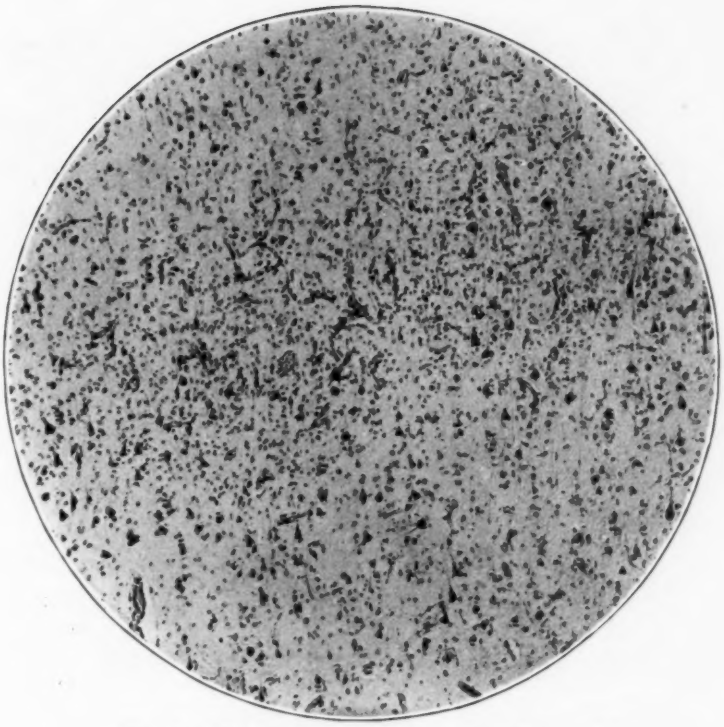

Fig. 2.—Higher magnification from Figure 1.

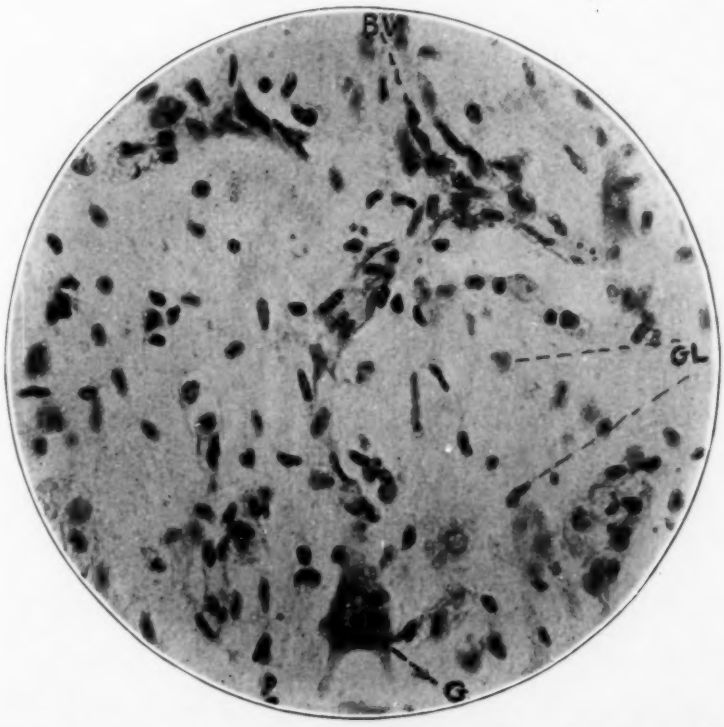

Fig. 3.—New vessel formation. Relative integrity of ganglion cell, *G*; altered glia, *Gl*; *BV*, blood vessel.

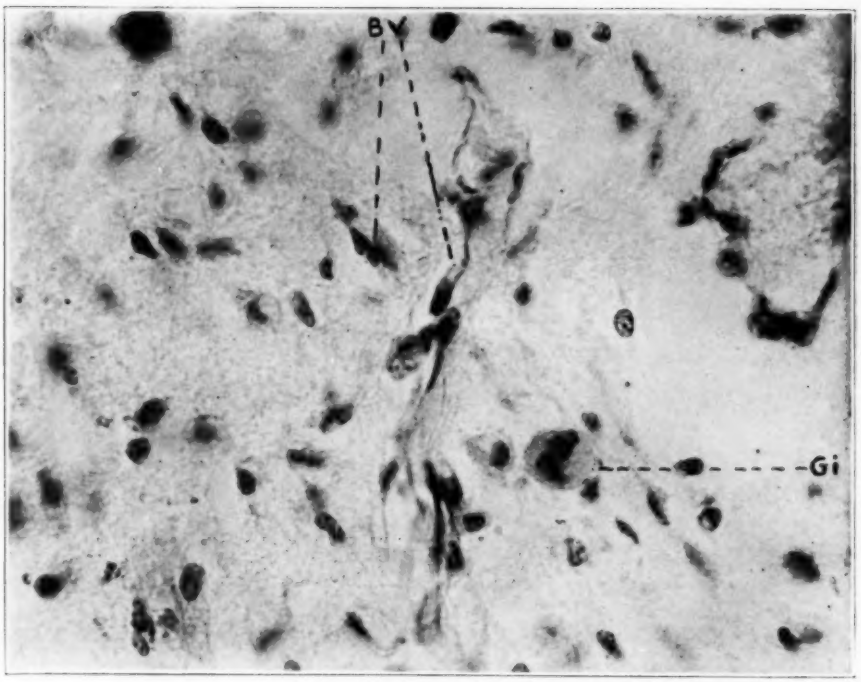

Fig. 4.—Magnification in oil of blood vessel showing budding. *BV*; *Gi*, gitter cell. Character of glia can be made out.

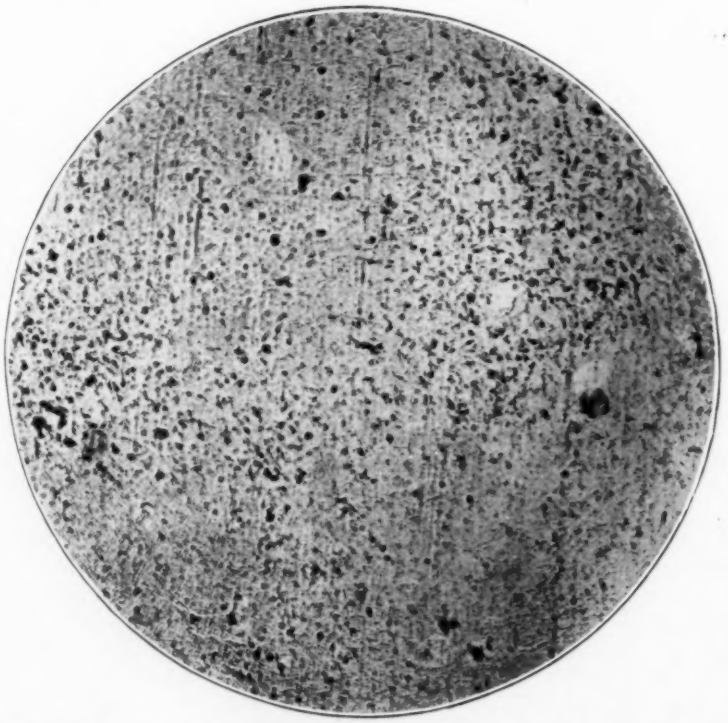

Fig. 5.—Sharlach R preparation showing numerous fat granules within affected area.

but was rather a selective cellular necrobiosis, since even in the center of this zone normal ganglion cells were present. It is proper to state, however, that many ganglion cells were undergoing destruction, that glia cells were increased, mainly of the compound granule type, and laden with fat droplets as seen in Herxheimer preparations (Fig. 5). The basal ganglia were but slightly involved and showed mainly acute cellular changes and not the softening that is usually found.

COMMENT

The whole picture differs from that which occurs as the result of vessel occlusion in that the area involved is too widespread throughout the cortex and too limited in its width to be in a vessel distribution. The minute pathology is also different. Many ganglion cells, normal in structure, remain; the vessels and glia are proliferated secondarily. Spielmeyer [3] has described, in experimental lead poisoning in a dog, a cortex that is absolutely identical, and he quotes Cerletti, who found vascular changes of a similar nature throughout the cortex in pernicious malaria.

The brunt of the pathology in this case was borne by the cortex in which there was an acute toxic condition limited to the third and fourth cortical layers, exactly similar to the case described by Stewart. Why this particular location suffered as the result of the poisoning is still in doubt. This phase is taken up by Stewart, who writes as follows:

> The areas with the most abundant blood supply are those in which most damage was sustained, and it appears probable, therefore, that a mere deprivation of oxygen could hardly have conditioned this peculiar distribution of cortical necrosis. On the other hand, if it be assumed that carbon monoxid acts like other exogenous poisons, the infragranular layers with their fine arterial meshes would be particularly exposed to its noxious effects. Moreover, experimentally produced anemia of the cortex, by ligation of the vertebral and carotid vessels, shows that the small and medium pyramids are more affected than the cells in the upper layers; the latter are, phylogenetically speaking, older and less liable to decay than are the more superficially placed cells. The observation that an impoverished blood supply leaves them relatively unaffected strengthens the view that an anoxemia alone could not have produced the peculiar zone of softening seen in this case.
>
> One other factor must be taken into consideration, the commonest cause of cortical softening is unquestionably arrest of the circulation either by thrombosis or embolism, which is favored in the gray matter by the anatomical arrangement and fine calibre of the venules and arterioles.

This explanation by Stewart sounds plausible, but we do not agree with him. No emboli or thrombi could be found in our case. It is known that the third cortical layer is the most vulnerable and hence may suffer first in any disease of the cortex. Just why it should be so

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3. Spielmeyer, W.: Histopathologie des Nervensystems, Berlin, Julius Springer **1**:227, 1922.

susceptible is not definitely known; the blood supply alone does not explain it. Spielmeyer [3] has described it in experimental lead poisoning; Cerletti (quoted by Spielmeyer) found it in pernicious malaria, and Jakob (quoted by C. and O. Vogt [4]) emphasizes it in spastic pseudosclerosis, dementia praecox and other conditions. The Vogts have given this particular suspectibility or vulnerability a special name of "pathoklise," which does not help to clear up the problem, but they believe that a physicochemical reason may account for peculiar locations of different disease processes.

Our patient lived seventeen days after exposure to gas, and she was unconscious all of this time, so that no accurate mental examination could be made. Had she recovered from the acute poisoning, she probably would have shown some degree of dementia or a psychosis because of involvement of the cortex. Indeed, it is likely that some of the patients that show mental changes (the commonest of which is amnesia) following carbon monoxid poisoning have a condition similar to that we have described.

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4. Jakob, in Vogt, C., and O.: Erkrankungen der Grosshirnrinde, J. f. Psychol. u. Neurol. **28**: (Sept.) 1922.

RELATION OF THE SYMPATHETIC NERVOUS SYSTEM TO MUSCLE TONE

EXPERIMENTAL AND CLINICAL OBSERVATIONS *

ALLEN B. KANAVEL, M.D.; LEWIS J. POLLOCK, M.D.
AND
LOYAL DAVIS, M.D.
CHICAGO

INTRODUCTION

A further impetus has been given to the surgical aspect of the sympathetic nervous system in a recent series of articles by Royle and Hunter.[1] Assuming as proved the principle that all striated muscle has a dual innervation from the cerebrospinal and sympathetic nervous systems, these investigators have predicated that increased muscle tonus, due to various types of lesions, may be decreased by interruption of the innervation of skeletal muscles from the sympathetic system. This

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* From the Departments of Surgery and Nervous and Mental Diseases, Northwestern University Medical School.

* The experimental work of this article is Contribution No. 108 from the Department of Anatomy of Northwestern University Medical School.

1. Royle, N. D.: A New Operative Procedure in the Treatment of Spastic Paralysis and Its Experimental Basis, Med. J. Australia **1**:77, 1924. Royle, N. D.: The Operations of Sympathetic Ramisection, Med. J. Australia **1**:587, 1924. Hunter, J. I.: The Postural Influence of the Sympathetic Innervation of Voluntary Muscle, Med. J. Australia **1**:86, 1924; On the Choice of Procedure Adopted in the Operation of Ramisection for Spastic Paralysis, ibid., p. 590; The Significance of the Double Innervation of Voluntary Muscle Illustrated by Reference to the Maintenance of the Posture of the Wing, ibid., p. 581.

subject is at once so interesting and so full of problems that it opens an unexplored field for clinical and experimental investigation.

Bearing in mind that the surgery of the sympathetic nervous system has tended toward clinical empiricism, we have thought that the relationship of the sympathetic system to the complex problem of muscle tone should be thoroughly investigated from as many angles as possible. It is only by establishing basic principles by clinical and experimental methods that our surgical therapeusis may be prevented from outdistancing our fundamental knowledge and thus escape misapplication.

In approaching this problem, several pertinent questions immediately suggest themselves. Is there definite evidence in favor of the dual innervation of striated muscle? What effect does sympathectomy have on muscle tone in the normal animal? Are there various clinical types of increased muscle tonus? What mechanisms in the central nervous system control muscle tone? What part do these mechanisms play in the production of abnormal muscle tone? What effect does the sympathetic system have on the production of various types of abnormal muscle tone? What is the effect of sympathectomy on decerebrate rigidity, which is the best example of experimentally produced increased muscle tone? Just how far and with what degree of success can we apply these theories to given clinical cases of muscular hypertonicity?

THE DUAL INNERVATION OF SKELETAL MUSCLE

The question of the presence of nonmyelinated motor nerve endings in skeletal muscles independent of the ordinary myelinated motor nerve fibers in structure, origin and connections has been contested for many years. As long ago as 1882, Bremer [2] described fine unmyelinated nerve fibers entering the motor end-plates in frogs and lizards. Some of these nonmedullated fibers had lost their myelin in their course in the muscle; some were branches of medullated fibers, but others arose from an unmyelinated plexus although they could not be traced further centrally. The two types of nerve endings were commonly, but not always, close together in the muscle fiber. Gerlach, Dogiel and Retzius contemporaneously were not able to record any specific distinction among the motor nerve endings.

Twenty years later, Perroncito [3] stated that striated muscle fibers were supplied with nerve endings from both the sympathetic and from the cerebrospinal systems. He described unmyelinated nerve fibers in voluntary muscles, but his descriptions and plates showed inconclusive proof of differentiation from ordinary unmyelinated filamentous continuations of the neurofibrillae of the motor end-plates. At about the

2. Bremer: Arch. f. mikr. Anat. **21**:165, 1882.
3. Perroncito, quoted by Mosso: Arch. ital. de biol. **41**:183, 1904.

same time, Grabower [4] examined the striated muscle fibers of man and was wholly unable to recognize any clear-cut distinction between the nerve fiber endings. He found many gradations from fine pale fibers to thick, dark medullated ones. Rarely did he see an unmyelinated fiber which could be traced for any great distance.

Interest in this subject then lagged until 1910 when Boeke [5] again brought it to the foreground. Subsequently, this author, with Dusser de Barenne,[6] de Boer [7] and others, have made important contributions to our knowledge of the subject. Boeke described nonmyelinated fibers which apparently were independent of the medullated fibers, and which ended in characteristic rings, loops and nets, either within the sarcoplasm or a typical end-plate or as small independent end-plates. He emphasized their hypoterminal position and their intimate association with the ordinary end-plates. At this same time, Botezat [8] described a dual innervation of the striated muscles of birds, but did not determine the derivation or function of the additional fibers. In 1913, in an effort to correlate his findings with those of previous workers, Boeke [9] stated that those unmyelinated fiber endings described by others were collaterals of medullated fibers and were entirely different from his "accessory" or unmyelinated fibers, which he stated were of sympathetic origin. In 1916, Boeke [10] resected the nerves supplying the eye muscles close to the brain. After from three to five days, using the Bielschowsky silver method, he found that all of the medullated fibers had degenerated, as had the end-organs associated with them. The "accessory" or unmyelinated fibers were intact, and he concluded that each muscle fiber

4. Grabower: Ueber Nervenendigungen in Menschlichen Muskel, Arch. f. mikr. Anat. **60**:1, 1902.

5. Boeke, J.: Die Motorische Endplatte bei den höheren Vertebraten ihre Entwickelung Form und Zusammendhang mit der Muskel faser, Anat. Anz. **35**:193, 1910; Beitrage zur Kenntnis der Motorischen Nervenendigungen, Internat. Monatschr. f. Anat. u. Physiol. **28**:377, 1911.

6. Barenne, J. G. Dusser: Ueber die Innervation und der Tonus der quergestreiften Muskeln, Arch. f. d. ges. Physiol. **166**:145, 1916; Once More the Innervations and the Tonus of Striped Muscles, König. Akad. v. Wetensch. **21**:1238, 1919; Ueber die Enthirnungsstarre in ihrer Beziehung zur efferenter Innervation der quergestreiften Muskeln, Folia neuro-biol. **7**:651, 1923.

7. De Boer, S.: Die Bedeutung der tonischen Innervation für die Funktion der Quergestreiften Muskeln, Ztschr. f. Biol. **65**:239, 1915; Die autonome Innervation des Skelett muskeltonus, Arch. f. d. ges. Physiol. **190**:41, 1921.

8. Botezat, E.: Fasern und Endplatten von Nerven zweiter; Art an den gestreiften Muskeln der Vögel, Anat. Anz. **35**:396, 1910.

9. Boeke, J.: Die doppette (motorische und sympatische) efferente Innervation der Quergestreiftan Muskeln, Anat. Anz. **44**:343, 1913.

10. Boeke, J.: Studien zur Nervenregeneration, Verhandl. d. k. Akad. v. Wetensch. **18**:91 and 19; No. 5, 171, 1916.

possessed at least one "accessory" end-platelet which came to it by way of the carotid sympathetic plexus. In a paper a year later, he showed that three weeks after section of the same group of nerves, the majority of these unmyelinated fibers had degenerated. He concluded, therefore, that some of these unmyelinated fibers were derived from the craniosacral group of sympathetic fibers, while the smaller number which resisted degeneration a longer time originated from the thoracolumbar outflow.

Boeke and Barenne [11] then worked on this problem together, and after section and degeneration of the anterior spinal roots and excision of the spinal ganglia of four consecutive thoracic segments, found that the nonmyelinated nerve fibers with hypoterminal endings in the intercostal muscles were intact. They concluded that these fibers must, therefore, be postganglionic sympathetic fibers. In this article, completely ignoring the results of his experiments on the motor nerves of the extraocular muscles, Boeke stated that these unmyelinated or "accessory" nerve endings remained unaltered after section of these nerves, and therefore they must be of sympathetic origin. Agduhr [12] removed the stellate ganglion in cats, and later found the remains of degenerating unmyelinated fibers in the muscles. In other cats, he resected the posterior roots and spinal ganglia of the last four cervical and first two thoracic communicans. After from five to ten days, the Bielschowsky method showed degeneration of all myelinated fibers and intact unmyelinated fibers.

As Langley [13] has stated, such unmyelinated nerve endings have been shown only by a silver impregnation method, and if all striated muscles contain them, it is to be remarked that they have never been seen in methylene blue preparations of the frog's sartorious muscle in which a certain number of somatic nerve endings and nerve fibers about blood vessels are easily and definitely brought out. In a recent and complete review of the question, Adrian [14] concluded that it should be accepted that voluntary muscle has a double nerve supply of medullated fibers from the anterior horns and nonmedullated fibers from the sympathetic ganglia.

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11. Boeke, J., and de Barenne, J. G. Dusser: The Sympathetic Innervation of the Cross-Striated Muscle Fibers of Vertebrates, Verhandl. d. k. Akad. v. Wetensch. **21**:1229, 1919; The Innervation of Striped Muscle-Fibers and Langley's Receptive Substance, Brain **44**:1, 1921.

12. Agduhr, E.: Sympathetic Innervation of the Muscles of the Extremities, Verhandl. d. k. Akad. v. Wetensch. **21**:930, 1919.

13. Langley, J. N.: The Autonomic Nervous System, Part I, Cambridge, W. Heffer & Sons, 1921.

14. Adrian, E. D.: Muscle Tonus and the Sympathetic System, M. Sc. Abstr. & Rev. **2**:454, 1920.

Therefore, in spite of contradictory facts, and from purely histologic evidence based on studies of sections stained by the silver impregnation methods, it is probable that all striated muscle contains two types of nerve fiber endings. The larger medullated fibers are those derived from the cerebrospinal system and constitute the ordinary motor end-plate fibers. The smaller unmyelinated fibers, which are independent of the former fibers and which end in rings, loops or nets either within the sarcoplasm of a typical end-plate or as small independent end-plates, are derived from the sympathetic nervous system. These facts, however, afford no conclusive evidence of the function of such fibers, and we can assume their functional activity only from the fact of their existence.

THE RELATION OF THE SYMPATHETIC SYSTEM TO MUSCLE TONUS

Mainly on the basis of Perroncito's work, Mosso [15] first suggested that the sympathetic system, from which the unmyelinated fibers originate, governed muscle tone and slow muscular contraction, while the cerebrospinal system, which gave origin to the myelinated fibers, controlled the rapid contraction of muscle fibers. These histologic descriptions also led de Boer to attempt to determine the function of the unmyelinated fibers which innervate striated muscles. He worked on the frog, and in 1915 produced evidence which tended to show that the tonic innervation of muscles was conducted from the spinal cord through the sympathetic system.

Langellan [16] at this time stated that tonic contraction and the phenomena allied with it were due to the sarcoplastic part of the skeletal muscle and that plasticity was the chief property of the sarcoplasm. He therefore stated that maintenance of a slight state of contraction was due to that element. He proposed to divide muscle tonus into contractile and plastic tone; the former innervated by the cerebrospinal motor end-plates, and the latter by sympathetic nerve endings.

Barenne,[17] in 1916, divided the abdominal sympathetic trunk in cats and frogs, and stated that there resulted a decrease in muscle tonus of the corresponding leg. He emphasized, however, that the loss was partial, was recovered from, and he did not believe that tonus in striated muscles was of sympathetic origin. About three years later, Barenne renewed his search for evidence of the influence of the sympathetic innervation of skeletal muscles on muscle contraction. He could find no clear evidence that the sympathetic system produced mechanical tonus, but thought it probable that it might influence chemical tonus.

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15. Mosso, A.: Theorie dela tonicité musculaire baseé sur la double innervation des umscles striés, Arch. ital. de biol. **41**:183, 1904.

16. Langellan, J. W.: On Muscle Tonus, Brain **38**:235, 1915.

17. Barenne: Footnote 6.

Kuno [18] carefully repeated de Boer's experiments, and found that division of the rami communicantes on one side in twenty-two frogs did not produce any change in muscle tonus. On the other hand, division of the anterior roots was always marked by relaxation of the muscles. Muscles which were still connected with the central nervous system showed, on adding a load, a gradual increase in length for from thirty to forty minutes. Kuno found that ramisectomy did not in any way alter this rate of stretching. Cobb [19] found no loss of tone in the hind legs and tail of cats on section of the sympathetic chain between the fourth and fifth lumbar ganglia. Saleck and Weitbrecht [20] sectioned the rami communicantes to the crural nerves, and found that there was no diminution in muscle tonus. They concluded in general that the tonic innervation to muscles of the extremities was not connected with the preservation of their sympathetic nerves. Kahn [21] found that the clasp reflex of frogs was accompanied by a tetanic action current and that the pathway of the efferent impulses was in the spinal nerves. Section of the sympathetic trunks was without any effect on this reflex. Kuré [22] and his co-workers believed that section of the sympathetic fibers, or of the phrenic nerve, produced a slight loss of tone in the diaphragm from which there was recovery. Cutting both of these nerves caused a marked and permanent loss of diaphragmatic tonus.

In 1922, Manmary [23] found that unilateral extirpation of the labyrinth and abdominal sympathectomy on the opposite side produced a loss of muscle tone only on the side of the divided sympathetic. Again reverting to experiments on the frog, Spiegel [24] found that removal of the sympathetic connections to the fore limb had no effect on the clasp reflex. He believed that muscle tone cannot be correlated with activity of the sympathetic fibers. Mansfield [25] criticized Barenne's work in particular, on the ground that division of the sympathetic trunks leads

18. Kuno, Y.: On the Alleged Influence of Adrenalin and of the Sympathetic Nervous System on the Tonus of Skeletal Muscle, J. Physiol. **49**:138, 1915.

19. Cobb, S.: A Note on the Supposed Relation of the Sympathetic Nerves to Decerebrate Rigidity, Muscle Tone and Tendon Reflexes, Am. J. Physiol. **46**:478, 1918.

20. Saleck, W., and Weitbrecht, E.: Zur Frage der Beteiligung sympatischen Nerven aus Tonus der Skelett Muskulatur, Ztschr. f. Biol. **71**:246, 1920.

21. Kahn: Arch. f. d. ges. Physiol. **177**:294, 1919.

22. Kuré, K.: Ueber den Zwerchfelltonus, Arch. f. d. ges. Physiol. **194**:577, 1922; Ueber den Zwerchfelltonus, ibid., p. 481.

23. Manmary, A.: Zur Frage der Abhängigkeit des Muskeltonus vom sympatischen Nervensystem, Ztschr. f. biol. **74**:299, 1922.

24. Spiegel, E. H.: Der Klammerreflex nach Sympatheticusexstirpation, Arch. f. d. ges. Physiol. **192**:115, 1921.

25. Mansfield, G.: Ueber die Innervation und den Tonus der Quergestreiften Muskeln, Arch. f. d. ges. Physiol. **168**:205, 1917.

to vasodilatation and increases the oxygen usage in the muscle. This would explain any transient relaxation which may occur.

This question of muscle tonus and its relation to the sympathetic system was investigated from still another angle which from its nature has proved instructive and interesting. Tonic contraction of the skeletal muscles is so well marked in decerebrate rigidity that we owe most of our knowledge of muscle tone to that preparation. The effect of sympathectomy on the muscular rigidity following decerebration therefore opened a fruitful field for investigation. Barenne,[26] Lopez and von Brücke,[27] von Rijnberk [28] and Cobb [19] all agreed that decerebrate rigidity is in no way dependent on sympathetic innervation. Rijnberk found no difference in the rigidity after sympathectomy in nineteen animals, and Cobb observed no difference in six cats.

Experiments have also been conducted to determine the effect of the sympathetic system on the use of oxygen and formation of carbonic acid in muscles. Mansfield and Lukacs [29] determined the respiratory exchange in dogs and found that in a lightly cruarized animal, section of the sciatic caused a decrease of respiratory exchange if the sympathetic was intact, but did not do so if the sympathetic had been cut. They maintained that sympathectomy abolished muscular tone and so decreased the respiratory exchange of the muscles.

Langley [13] has insisted that conclusive evidence is entirely lacking that any slight loss of muscle tone resulting from sympathectomy is not the result of section of vasomotor nerves and consequent vasodilatation. He has also pointed out that stimulation of the sympathetic trunk has no effect on striated muscle except to decrease its use of oxygen.

So the matter stood with proof that striated muscle fibers contained unmyelinated nerve endings of sympathetic origin; with extremely debatable evidence that sympathectomy produced a loss of tone in normal animals, and with entire agreement that section of the sympathetic had no effect on the production of muscular rigidity following decerebration. Then early in 1924 the subject was reopened by Royle, who had been studying the dysfunction and deformities produced by spastic paralysis. He conceived the idea of applying this mass of data to the clinical cases which to his mind presented disability in the main produced by hypertonus in antagonistic groups of muscles. Assuming that interruption of the sympathetic system would decrease muscle tonus, Royle operated on

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26. Barenne, J. G. D.: Folia neuro-biol. **7**:651, 1923.

27. Lopez, J., and von Brücke, E.: Nach der Bedeutung des Sympathicus für den Tonus der Skelettmuskulature, Arch. f. d. ges. Physiol. **166**:55, 1916.

28. Von Rijnberk: Recherches sur le tonus musculaire et son Innervation, Arch. de Physiol. **1**:702, 1916.

29. Mansfield, G., and Lukacs: Arch. f. d. ges. Physiol. **161**:467, 1915.

two patients, both of whom showed increased muscle tonus due to a tramuatic cerebral lesion. He divided the abdominal sympathetic trunk and sectioned the gray rami from the second, third and fourth lumbar ganglia in one instance, and sectioned the gray rami to the cervical nerves innervating the muscles of the upper extremity in another case. After operation in both instances, the affected limb was warmer, but there was no difference detected by the use of a surface thermometer. The extremity became brighter in color, with distinct evidence of vasodilatation, which had disappeared in one instance by the seventeenth day. The decrease in muscle tonus was noticeable immediately after the patient awakened from the anesthetic, and the muscles became more relaxed as time went on. The patients are reported to have been able to perform many acts following the operation, which previously were difficult or wholly impossible. Although no report is available in the literature, it is said that attention has been directed toward the relief of the muscular rigidity in extrapyramidal lesions, such as paralysis agitans both of the true type and that following epidemic encephalitis, with less satisfactory results.

In order to clarify the situation from an experimental standpoint, Royle in conjunction with Hunter has sectioned the sympathetic rami communicans in goats and in fowls. They state that there is immediate decrease in muscle tone, which persists. If goats, so operated on, are placed on their backs, the sympathectomized extremity will assume a much less tonic posture than the opposite normal leg. Hunter has cut the anterior spinal roots supplying the muscles of the wings of fowls and has obtained a definite flaccid posture. Later, in the same animal, he removed the sympathetic innervation to the same extremity. This resulted in a further and more complete loss of postural tone. It seems quite possible to explain the results of such experiments on the fowl on the fact that in such heavily feathered animals, the arrectores pilorum muscles are well developed. They perform the specialized function of ruffling the feathers and hence must contribute an important part to the posture of the wing. Their innervation from the sympathetic system is well known, and the diminution in development and functional importance through the animal scale to man is of importance. It would seem that such an experiment would have been much more convincing had the feathers been plucked.

Royle and Hunter have studied a series of experiments on goats to determine the effect of ramisectomy both before and after decerebration. The guillotine method of decerebration was used throughout their experiments. Contrary to all the former work, Royle and Hunter obtained a difference in the degree of extensor rigidity on the affected side. They have stated that the onset of decerebrate rigidity occurs as

it does normally but is not maintained in the affected limb. They emphasize the fact that the longer the interval between division of the sympathetic system and decerebration, the more pronounced is the difference in the degree of rigidity assumed. They criticize previous workers by stating that insufficient time had elapsed between ramisectomy and decerebration. It should be noted, however, that clinically the muscular relaxation is said to have been immediate. In discussing their results, Royle and Hunter state that in spasticity the two components of muscle tone are increased. Increased contractile tonus leads to hyperactive tendon reflexes and confers posture on the extremity. Increased plastic tone leads to permanent maintenance of that imposed posture, from which the limb cannot free itself.

It is perfectly evident that the effect of removal of the sympathetic influence on the experimental production of decerebrate rigidity and on the normal tone of striated muscles must be conclusively established in one direction or another before any logical interpretation can be given to clinical results. Before proceeding, therefore, to the results obtained in our clinical cases, we wish to state the results of our experimental observations.

EXPERIMENTAL OBSERVATIONS

In all of our experimental work, cats were used as the animals of choice. Such animals were chosen because in addition to the fact that they are common laboratory animals, they have been used from the beginning by Sherrington and others to study muscle tone in all of its phases. Further, the cervical and abdominal sympathetic trunks are easily exposed in cats without the production of trauma which might interfere with the interpretation of results. Moreover, cats may be decerebrated with extremely satisfactory results either by the guillotine method or by the method of ligature of the basilar and carotid arteries.

Eighteen animals form the basis for our studies. Sixteen animals that had been operated on by ourselves and two that had been operated on by another were studied. Of these animals, the right cervical sympathetic chain, including the stellate ganglion, was removed in six. The right abdominal sympathetic trunk was removed from the second lumbar ganglion to and including the fourth lumbar ganglion in six animals, and the abdominal trunks on both sides were removed in the other four. The right stellate ganglion alone was removed in the two animals which were operated on by Sarah Towers of the physiology department, and which we had the privilege of observing.

These animals were studied after they had recovered from the anesthesia and at frequent intervals as long as from two weeks to sixty-five days after operation. The tendon reflexes, active and passive motion, posture of the limbs and coordinated movements were studied in addition

to the vasomotor and other changes incident to the removal of the sympathetic chain. Motion pictures were taken of these animals in various postures and in walking, running and jumping.

In none of the animals were we able to record any changes in the character of the deep tendon reflexes after removal of the sympathetic innervation to an extremity. The response to stimulation and the fall of the limb were the same on the operated and normal extremities.

In studying active movements of the extremities, the animals were made to stand to reach for food and to strike at a moving string with the forelimbs, as a cat at play will do. They were made to run about the laboratory to determine whether or not fatigue would bring out a limp or any other change in muscle tone. Further, the animals were pushed from high tables, and their manner of striking the floor was observed. By none of such methods were we able to observe or to record by the motion camera any difference in the extremities. Several unprejudiced observers were unable to detect any changes in the limbs.

These animals were then placed on their backs with the weight of the animal exactly in the midline. In such a posture, a normal animal will keep all four extremities in extension. If, however, the head or trunk be slightly moved in one direction or another, the extremities on the side to which the chin points tend to assume an increased attitude of extension, while those on the opposite side become flexed. These facts are then of considerable importance in studying limb postures. The animals were suspended by the skin of the neck and lumbar regions, and the posture and resistance of their limbs to passive motions were observed. With the animals in any of these positions we were unable to detect any difference between the normal and affected limbs.

Twelve of the cats so operated on were decerebrated by a method described by two of us.[30] Briefly, this consists in ligating the basilar artery at the level of the exit of the fifth cranial nerves from the pons, and ligating both common carotid arteries in the neck. Such a method produces anemia cephalad to the level of the ligature on the basilar artery. The level of decerebration can be easily verified subsequently by the injection of methylene blue into the circulation. Animals so operated on immediately assume the characteristics of a decerebrate preparation. Moreover, they have pinna, corneal, scratch, sneeze, and extensor thrust reflexes which can be demonstrated immediately. They therefore are decerebrate animals which are much more favorable for study than are those produced by the comparatively crude, rough guillotine method, which is accompanied by severe hemorrhage and shock, and in which the level of decerebration cannot always be accurately

30. Pollock, L. J., and Davis, L. E.: Studies in Decerebration. I. A Method of Decerebration, Arch. Neurol. & Psychiat. **10**:391 (Oct.) 1923.

determined. Further, animals decerebrated by such a method can be kept alive without difficulty as long as forty-eight hours, even though no nursing care be given them. That such animals are not products of midbrain irritation is easily proved by the fact that they show Magnus de Kleijn phenomena; by the fact that they can be kept in such a state at least for forty-eight hours, whereas anemia of the central nervous system for longer than fifteen minutes produces a change in the tissues from which the animals cannot recover. Decerebration was performed on these animals at periods following the removal of the sympathetic innervation, varying from fourteen to sixty-five days. They consequently meet the time element emphasized by Royle and Hunter.

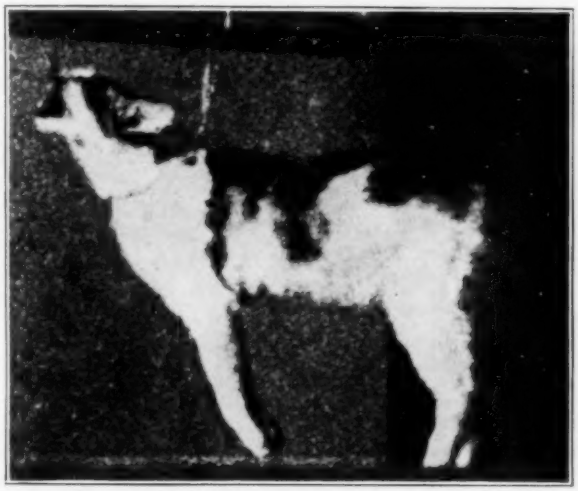

Fig. 1.—Equal rigidity in forelegs of decerebrate cat thirty-two days after a right cervical sympathectomy.

The typical decerebrate animal is one in which transection has been produced at any point between the anterior corpora quadrigemina and the calamus scriptorius. In decerebrate rigidity, all the muscles which keep the animal in the standing position are in strong tonic spasm. The extensors of the limbs, the extensors of the spinal column and the elevators of the head, neck and tail are in strong tonic spasm. On being suspended, the decerebrate animal is seen to have its forelimbs stiffly thrust backward with retraction at the shoulder joint, straightened elbow and slight flexion at the wrist joint. The hind limbs are similarly straightened and thrust backward. When the limbs are moved from the position they have assumed, considerable muscular resistance can be palpated. On sudden release, they spring back to their original position more rigid than before. The muscular rigidity in such animals is quite

plastic. The limbs tend to retain a new length imposed on them either by passive manipulation or by eliciting reflex movements. In other words, they show shortening and lengthening reactions. It will be realized, therefore, that the decerebrate animal, as emphasized by Royle and Hunter, is a favorable preparation on which to study the effect of removal of the sympathetic supply to a limb.

In all of our animals so decerebrated, the onset of decerebrate rigidity and its maintenance was exactly similar in all of the extremities (Fig. 1). This was true when the animal was studied lying on its back or suspended by its neck and back. In several of our decerebrate animals, we were able to produce running movements, which break through the existing increased extensor tone. This would seem to be one of the most

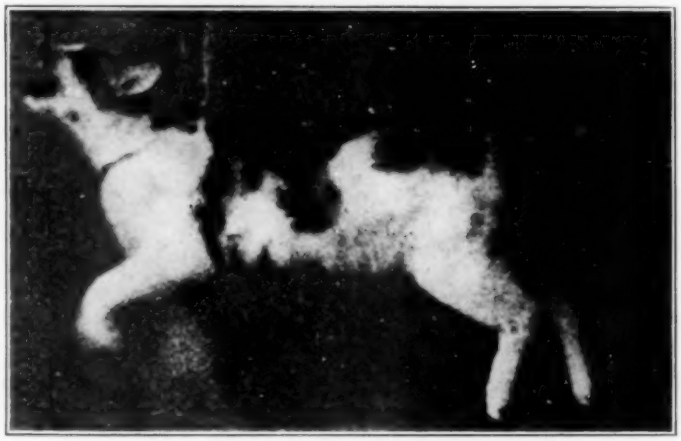

Fig. 2.—Running movements in decerebrate cat after cervical sympathectomy on right side. Both forelegs are used equally well.

favorable conditions in decerebrate rigidity in which to study any difference in the normal and affected extremities. Such a difference could not be observed, nor could it be recorded by a motion picture camera (Fig. 2). Resistance to the extensor rigidity was equal in the operated and normal limbs (Fig. 3).

We are forced to conclude, therefore, that the removal of the sympathetic supply to the limbs of cats has no appreciable effect on the normal muscle tonus in that extremity. Further, that the removal of the sympathetic supply to the limb has no effect on the onset, maintenance, or any of the characteristics of decerebrate rigidity, even though rather long intervals be allowed to elapse between the removal of the sympathetic supply and the decerebration.

CLINICAL EXPERIMENTS [31]

The question of muscle tone is a baffling one. Unequipped with an accurate conception of the physiology of tone, there is at present no satisfactory and definite clinical method of measuring changes in muscle tone. Much less is there a satisfactory means of determining small changes between two types of muscle tone. Realizing that such difficulties were present, we nevertheless attempted to produce graphic records of variations which might occur after the removal of the sympathetic supply to a limb. Such graphic tracings afford a definite record and seem to us to be indispensable in corroborating or disproving clinical observations, regardless of how many of the latter are made or who makes them. To depend alone on statements of patients or even on careful descriptions made by those interested in the problem, seems to us to be untrustworthy.

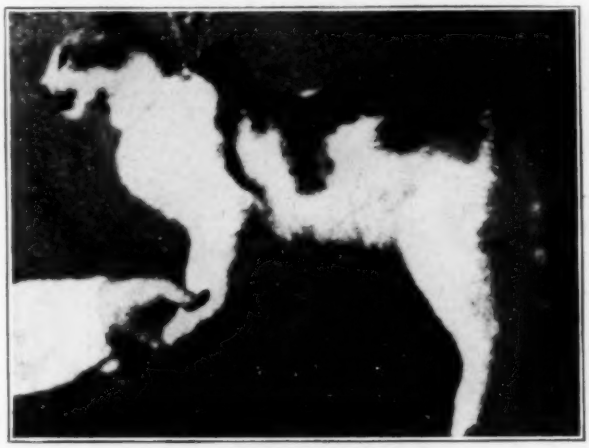

Fig. 3.—Attempt to break rigidity in forelegs of decerebrate cat after cervical sympathectomy on the right side.

We have, therefore, studied each of the cases in which operation was performed, as carefully as possible, before and after operation. Such studies have included the kymographic tracings of active and passive motion, tendon reflexes, tremors, responses to faradic stimulation, direct myotatic irritability, and active and passive movements after atropinization in some of the cases. We have also taken electromyographic tracings of tremors and active and passive movements. We have made complete roentgen-ray studies to determine the effect of interruption of the abdominal sympathetic trunk on the movements of the gastro-

31. A complete detailed record of the cases in which operation was performed will be given in a paper to be published in the near future.

intestinal tract. In addition to these methods of investigation, we have made motion pictures of the gait, range of active movements, resistance to passive movements and reflexes of the patients. As a matter of course, each of us recorded our personal observations independently.

At the beginning of this work, Royle's indications for operation were not available in the literature. We, therefore, chose examples of various clinical types of hypertonicity. The patients operated on thus far have included those with cases of postencephalitic Parkinson's disease, true paralysis agitans, lateral sclerosis of the spinal cord, cerebral hemiplegia with aphasia, and Little's disease and spastic paraplegia in extension

Fig. 4.—Attempt to move left arm before operation. Note tremor (postencephalitic Parkinson's syndrome).

due to a traumatic lesion of the spinal cord. All of the patients operated on have had a mentality and sufficient cortical control to make their reeducation possible. The patients with Little's disease, two of whom were paraplegic and the third hemiplegic, were attending school and were showing satisfactory progress. All of the patients exhibited shortening and lengthening reactions on reflex stimulation. In other words, the extremities would assume a new posture imposed on them. The tendon reflexes showed a "hung-up" characteristic; some patients, of course, exhibited this more than others. It will be recognized that these latter

characteristics are those exhibited by the decerebrate animal, in which they have been assumed to be representative of an increase in plastic tone. It is, of course, manifestly impossible to see the typical decerebrate preparation commonly duplicated in the clinic. Only two such clinical cases are on record. On the other hand, we believe, as does Walshe, that patients with spastic hemiplegia and paraplegia in extension show fragments of the picture seen in decerebrate rigidity, such as shortening and lengthening reactions and modification of posture by reflex stimulation.

In our operations on these patients, we have employed several means of approach. We have removed the entire cervical sympathetic trunk

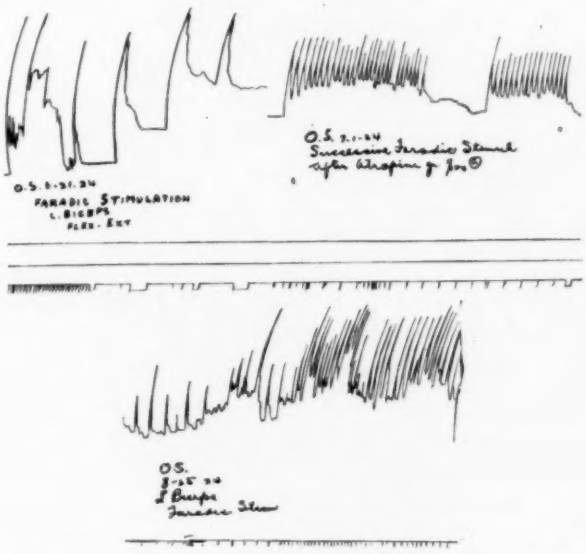

Fig. 5.—Kymographic tracing of response of left biceps muscle to faradic stimulation before operation, after atropinization and after operation (post-encephalitic Parkinson's syndrome).

and its ganglia, including the stellate ganglion; we have removed the latter alone; and we have removed the abdominal sympathetic trunk on one or both sides from the second lumbar ganglion to and including the fourth lumbar ganglion which lies over the brim of the pelvis. We have performed the latter operation both by the lumbar approach advocated by Royle and through a midline abdominal incision.[32] All of the rami communicantes, of course, must be severed in removing the sympathetic trunks, and it will be seen that approaching the operative procedure

32. The complete description of the operative technic will be given in a future publication.

from the side of the trunk rather than by avulsion of the rami would avoid any chance of trauma to the corresponding spinal nerves. It will be recognized that this is an important matter in dealing with spasticity in an upper extremity, since the slightest trauma to the brachial plexus will produce immediate temporary flaccidity. We feel that an abdominal approach to the sympathetic trunk is of distinct advantage if one is required to remove both abdominal trunks. In unilateral cases, we believe the lumbar approach to be the operation of choice.

In order that we might properly evaluate the effects of sympathectomy, we purposely refrained at all times from suggesting to the patient that he might be improved. We did not institute physiotherapy until reasonable time had elapsed after operation so that any results obtained could be ascribed alone to the surgical proceedure. This is recognized as an important factor in drawing conclusions, since remarkable improve-

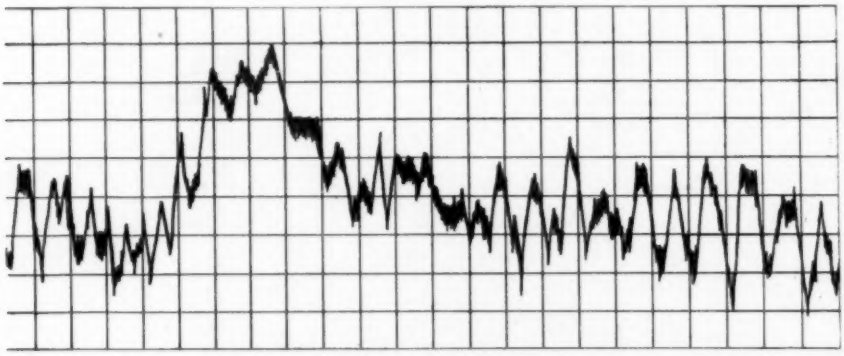

Fig. 6.—Electromyographic tracing of tremor of left upper extremity following operation (postencephalitic Parkinson syndrome).

ments in these cases have been obtained by others by the use of reeducational physiotherapy alone. We realize that many of these patients have long standing bony and fibrotic changes which necessarily interfere with the performance of movements, and such changes were always taken into consideration.

Postencephalitic Parkinson's Disease.—After cervical sympathectomy on one upper extremity and bilateral abdominal sympathectomy, we are unable to record any improvement in the character of the tremor, the gait, the range or smoothness of active movements, or in the resistance to passive motion. Figure 4 is the motion picture photograph of the patient before operation and shows the attempt to perform active movements in the upper extremity. Figure 5 is a kymographic tracing which shows the response to faradic stimulation before and after operation and after atropinization. It will be seen that atropin effects a marked

improvement in the character of the muscular response, which was not obtained by the operation. Figure 6 is an electromyographic record of the patient's tremor and shows no change after operation. Gastrointestinal roentgen-ray studies three months after operation revealed no changes in the peristaltic movements. There was no roentgen-ray evidence of megacolon, and the patient had no symptoms of either diarrhea or constipation.

Lateral Sclerosis.—Figure 7 shows the attempts of this patient to move actively his lower extremities before operation. There was no appreciable change in the character of the movements after bilateral abdominal sympathectomy. This is shown graphically in Figures 8 and 9. Resistance to passive movements of the legs was marked, and this remained unchanged. The responses of the quadriceps muscles to

Fig. 7.—Attempts of patient to move lower extremities before operation (lateral sclerosis).

faradic stimulation showed characteristic steplike ascension, as did the tendon reflexes, and this characteristic has remained unchanged four months after operation.

Cerebral Hemiplegia with Aphasia.—These cases exhibited typical "hung-up" reflexes and lengthening and shortening reactions before operation. All were patients with right-sided hemiplegia, with marked resistance to passive motion. Figure 10 is a kymographic tracing of the right knee reflex after operation, and may be compared with that of the normal left knee reflex. This is a typical example of "hung-up" reflex, though a sympathectomy had been performed. A reduction in the plastic element of muscle tone should of course tend to obliterate such a steplike ascent, which is typical of a shortening reaction. Figure 11 is a motion picture photograph showing the range of movement possible in

the right arm before operation, while Figure 12 shows the same movement after operation. We felt that there was some improvement in the character of the movements after a cervical sympathectomy, but kymographic tracings (Figs. 13 and 14) show the tracing characteristics of flexion and extension in the right biceps muscle to be unchanged. Figure 15 is a motion picture photograph after several weeks of physiotherapy, and illustrates the improvement in attempts to balance on the affected limb. This is a feat which was entirely impossible before operation.

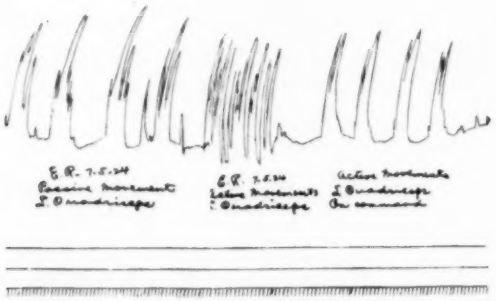

Fig. 8.—Kymographic tracing of active and passive motion in left quadriceps before operation (lateral sclerosis).

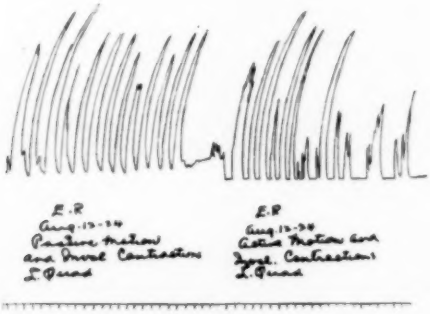

Fig. 9.—Kymographic tracing of active and passive motion in left quadriceps after operation (lateral sclerosis).

Paralysis Agitans.—The results obtained in true paralysis agitans have been exactly comparable to those described in the postencephalitic type of this disease. We have been wholly unable to record any change in the character of the tremor or in the ease of performance of active movements. The resistance to passive movements was of the type commonly found in this disease, and after three months we have been unable to record any improvement.

Little's Disease.—We have studied three patients with this type of case, all of whom were mentally alert. Two had spastic paraplegia and

one was a hemiplegic. All had been operated on to correct various orthopedic deformities, and all had been trained by physiotherapeutic measures. Figure 16 is the photograph of one of the paraplegic patients before operation, and shows his scissors-like gait. Figure 17 is a kymographic tracing which compares the knee reflex of the limb previously operated on with that of the opposite affected extremity on which operation had not been performed. The characteristic "hung-up" type of reflex may still be seen to be present. Figure 18 is a tracing of active motion in the right leg after operation. It will be noted that the down line which represents flexion is a gradual falling, wavy line which denotes the rigidity still present in the flexor muscles. Careful

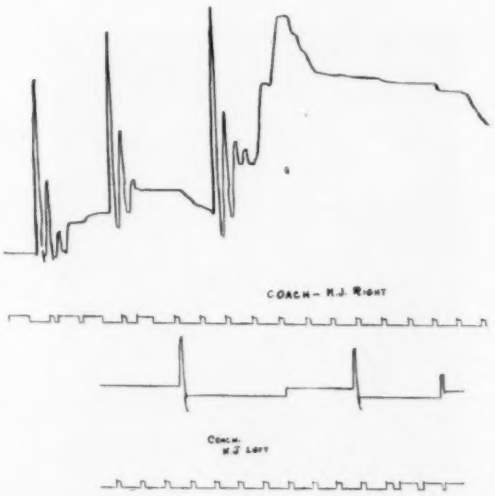

Fig. 10.—Kymographic tracings of right and left knee reflexes after operation. Note "hung-up" character of right knee reflex (right hemiplegia with aphasia).

observations on the resistance to passive motion, the rapidity with which the extremities fell from an imposed position, the position of the toes and the range of active motion have to date shown no striking changes.

Traumatic Spinal Cord Lesion.—A patient with spastic paraplegia in extension due to a gunshot wound of the spinal cord was also studied. The dysfunction in this case was preeminently that of rigidity and not of paralysis. The knee reflexes were characteristically "hung-up." This shortening reaction is shown in Figure 19. After resection of the abdominal sympathetic trunk on one side, no change in the character of the knee reflex or in the resistance to passive motion has been recorded. The range and facility of active motion remains unaltered.

Attention must be called to the fact that immediately after interruption of the sympathetic supply to a limb, there is definite increase in warmth of the extremity. Although this is difficult to measure with a thermometer, it can be detected easily. In addition, the capillary bed is markedly dilated, and the extremity has a suffused and flushed appearance. These changes have disappeared on the average of from five to six days after operation. There was distinct decrease in the secretion of sweat on the side of the face corresponding to the removal of the cervical sympathetic trunk in the cases of paralysis agitans. A typical Horner's syndrome accompanied the removal of the cervical sympathetic trunk in each instance. No alterations in the cardiac rhythm or rate were observed.

Fig. 11.—Range of motion in right upper extremity before operation (hemiplegia).

COMMENT

Tone is a property of muscle, the components of which are today incompletely known and the physiology of which is poorly understood.

Increase of tone, at first attributed solely to interruption of the corticospinal tract with heightened spinal reflexes, is now recognized to be the result of many conditions, some of which may be proved to exist, but most of which are conjectural.

Clinically it is known that hypertonicity occurs as the result of corticospinal tract lesions. Second, it occurs in a different form in the so-called extrapyramidal motor diseases, such as paralysis agitans and

other diseases of the striate body. Third, increase of tone is present in rare cases of complete decerebrate rigidity in man. Fourth, in certain diseases which probably involve a peripheral muscular mechanism, such as myotonia and tetany, hypertonicity is found. Lastly, tone is increased in a modified and temporary manner in certain frontal lobe lesions which supposedly involve a hypothetical cortical representation of tone.

Whether or not pure lesions of the corticospinal tracts produce hypertonicity as the result of increased spinal reflex activity, as expressed solely by increase of deep reflexes, is by no means assured. It is significant that certain cases of hemiplegia present increased deep tendon reflexes several weeks following apoplexy and yet display definite

Fig. 12.—Range of motion in right upper extremity after operation (hemiplegia).

diminution of muscle tone. Then, too, in early infancy, before myelinization of the pyramidal tracts is completed, muscle tone is diminished while the tendon reflexes are lively.

The increase of tone produced by so-called extrapyramidal disease is quite different from that seen in lesions which for the most part involve the pyramidal tracts. It does not exert any selectivity for flexors or extensors but affects all muscle groups more or less equally. Such hypertonicity is not associated with increased deep reflexes nor with that plasticity characterized by lengthening and shortening reactions. Neither can this type of hypertonicity be modified by certain phasic spinal reflexes.

Rarely hypertonicity is seen in certain gross lesions of the midbrain and pons as the result of complete or almost complete decerebration. It must be emphasized that decerebrate rigidity, as described by Sherring-

ton,[33] is a clearly defined condition. The decerebrate animal is one in which the brain stem has been divided between the level of the entrance of the eighth pair of cranial nerves and a plane bounded dorsally by the caudal border of the anterior colliculi and ventrally by the corpora mamillaria. In such a condition the extensor or antigravity muscles enter into a state of heightened tone which maintains the animal in a standing posture. Sherrington [34] found that although the tone of a given

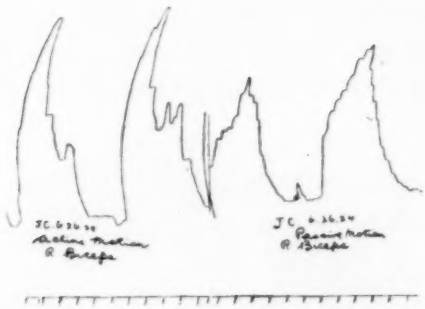

Fig. 13.—Kymographic tracings of active and passive movements in right biceps before operation (right hemiplegia with aphasia).

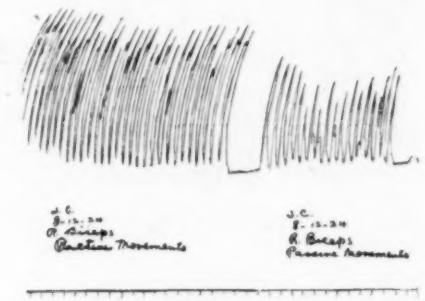

Fig. 14.—Kymographic tracings of active and passive movements of right biceps after operation. Speed of drum is increased over that in Figure 14 (right hemiplegia with aphasia).

muscle is dependent on intact afferent nerve supply, it may be modified by afferent impulses from other regions. Thus Magnus and de Kleijn have shown that modification of tone and therefore posture may be obtained through labyrinthine and neck reflexes. If, instead of the lesion described, both cerebral hemispheres are removed, and the

33. Sherrington, C. S.: The Integrative Action of the Nervous System, New Haven, Yale Press, 1906.

34. Sherrington, C. S.: On the Anatomical Constitution of Nerves of Skeletal Muscles, J. Physiol. **17**:211, 1895.

thalamus, subthalamic region and midbrain are left intact, the animal's reactions are different. The distribution of tone is normal, and no rigidity can be demonstrated. The animal can be stimulated to movement in various ways and the sum total of these reflex movements are designated by Magnus as position reflexes as opposed to the tonic reactions described in the former animal as standing reflexes.

When purely spinal animals have been prepared, numerous motor spinal integrations are still possible, such as the scratch reflex, extensor thrust and stepping movements.

Fig. 15.—Attempt of patient to balance on the right foot after operation (hemiplegia).

Thus it may be seen that there are integrative combinations of movement at various levels of the central nervous system. These are quite simple in the phylogenetic older and lower levels, such as the spinal cord, while they are more complicated at higher and more recently acquired levels. When all but the lowest spinal levels are removed, certain patterns of movement and tone are released. These primitive movements may be activated by higher levels, which when isolated from above permit certain specific postures which result from standing reflexes. In turn, if still higher levels be added to the lower ones and isolated from the cerebrum, position reflexes will result.

Clinically, certain phenomena found in decerebrate animals; namely, increased deep reflexes, shortening and lengthening reactions, and a

modification of tone by certain phasic spinal reflexes, are found in hemiplegia, cerebral diplegia of pyramidal tract origin and paraplegia in extension. However, these phenomena are often masked by increased spinal reflexes, contractures and muscular fibrosis. It is illogical to say that increase of tone in such cases is due to decerebrate rigidity. It may be said that some fibers are interrupted in both clinical cases and in decerebrate animals which produce the phenomena above mentioned. It may be admitted that there are at least two factors responsible for such spasticity; interruption of the corticospinal tract and interference with the functions of the brain stem mechanism. Further, we must not

Fig. 16.—Scissors-like gait of paraplegia before operation (Little's disease).

lose sight of the possibility of release of function from the cerebellum, postulated first by Hughlings Jackson and made probable by Tilney's work on the cocontraction function of the cerebellum. Such a release of function would undoubtedly produce a type of hypertonicity, although not related to decerebrate rigidity.

From the foregoing it may be seen that there are three mechanisms generally accepted as being concerned in hypertonicity: the corticospinal tract, the extrapyramidal system, represented largely by the striate body, and the brain stem mechanism. Finally, it would appear possible that a fourth could be postulated; namely, cerebellar cocontraction. It would likewise appear possible that there may be some peripheral mechanism of

muscle metabolism particularly active in such diseases as tetany, in which condition muscular cramps, changes in electric excitability and in irritability of muscles are found.

It has been suggested that that element of muscle tone which is related to the static or postural, in contradistinction to the kinetic, motor system, is mediated by the sympathetic nervous system through a high reflex arc in the pons and a lower one in the cord. The afferent and

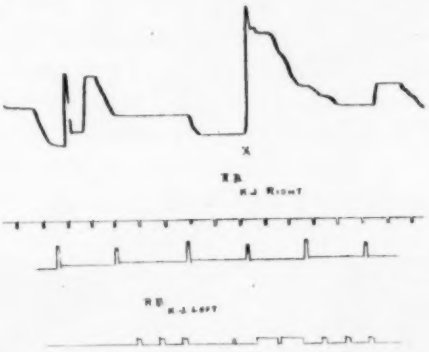

Fig. 17.—Kymographic tracings of right and left knee reflexes after operation on the right side (paraplegia—Little's disease). Note "hung-up" character of right knee reflex.

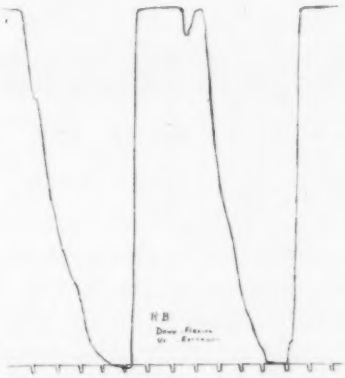

Fig. 18.—Kymographic tracing of active motion in right leg after operation. Note flexion movement as compared to extension (paraplegia—Little's disease).

efferent limbs of the lower arc would arise and terminate in the sarcoplasm of skeletal muscles. It would be highly unreasonable to assume that such a reflex pathway would serve all of the functional mechanisms previously mentioned. If it be true, sympathectomy should abolish that tone which in spasticity is due to hyperactivity of static or postural tone. This type of tone has been purported to be recognized by those characteristics which were described by Sherrington as the plastic tone

found in decerebrate animals and which, therefore, are present in all cases of hemiplegia, cerebral diplegia of pyramidal tract origin and paraplegia in extension, although often masked by other phenomena. It is certain that increased plastic tone, which is said to be modified by sympathetic ramisectomy, is not the only type of hypertonicity which is present in corticospinal tract lesions, cerebral diplegia, paraplegia in extension or in decerebrate rigidity. Certainly in decerebrate animals, leaving aside the question of plastic tone, the postural labyrinthine and neck reflexes are unaffected after sympathectomy. Although presenting the greatest degree of rigidity found in any experimental preparation, these animals are not well suited to the study of increased tone in its simplest form. The degree of rigor or retention of an imposed position is dependent on so many phasic reflexes that it is difficult to determine whether a difference in the rigidity of the extremities is due to the

Fig. 19.—Shows characteristic "hung-up" knee reflex before operation (traumatic spinal cord lesion).

improper development of the rigor or to artificially imposed changes. For example, if the animal lies on its side while the rigidity develops, the extremities on the dependent side will be less rigid and will be the first to relax as the result of phasic reflexes. It would seem to us that if this rigidity or fixation and retention of an imposed posture were due to a neural mechanism, as expressed purely by a reflex action, it would not vary as to degree and duration proportionate to the time allowed for its development. If a decerebrate animal be permitted to hang suspended and absolutely undisturbed for many hours, the rigidity becomes so marked that phasic reflexes no longer influence it, whereas if the position be changed or the animal be disturbed, the rigidity may be varied at will by phasic reflexes.

Further, we are unable to record any graphic changes in the condition of muscle tone in any of the clinical lesions mentioned above, following removal of the sympathetic innervation to an extremity. We

have also been unable to record or observe any changes in the hypertonicity produced by the release of function in straital disease or in traumatic or degenerative lesions of the spinal cord which have for the most part involved the lateral pyramidal tracts.

If hypertonicity is the result of removal of the inhibition of corticospinal influences alone or in combination with hypothetical cerebral sympathetic fibers, it seems strange that complete removal of the brain cephalad to the red nucleus does not produce hypertonicity in animals. If, on the other hand, hypertonicity is due alone to removal of the influences of the corticospinal tract and the upper levels of a sympathetic reflex arc, it is difficult to explain why complete section of the spinal cord fails to produce spasticity. It cannot be because of spinal shock or diaschisis because of the existence of mass reflexes in a so-called isolated segment of the cord. It is certain that a great amount of investigation is necessary to show why incomplete lesions do, and complete lesions, with the exception of those in the brain stem, do not produce hypertonicity. If there are a number of factors concerned with increase of tone, then it is necessary quantitatively to measure the diminution of tone, if any, which results from removal of the sympathetic nerve supply. This is necessary to obviate the inaccurate interpretation of any clinical improvement as due to lessened muscle tone, when there may be other factors involved. In our opinion, changes in muscle tone cannot be determined by the ordinary clinical examination, by the statements of patients or even by clinical results which have before been produced by hope and reeducation. It must be demonstrated by careful physiologic research and objective measurement.

It is possible that the sympathetic nervous system may have some function relating to the tone of muscles; or better, it may have some function dealing with the metabolism of muscles so that under certain conditions the contractility of a muscle may be changed by the removal of sympathetic impulses. Such a function may deal with colloidal chemistry. It may be possible that the increased vascularity which follows sympathectomy may influence the quality of muscle tone. It may be possible that because of modified metabolism or circulation the character of the afferent impulses from muscles is changed; as an example, the occurrence of pain in an anemic muscle. It may further be possible that certain apparent changes in range of motion may be due to vasodilatation in the synovial membrane and a concommitant increase in secretion of synovial fluid. Under any of these conditions, the changes produced by sympathectomy will probably be found to be temporary as are the vasomotor skin changes.

If, because of the otherwise hopeless character of spastic paralytics, it is decided that an operation for the purpose of removing sympathetic

impulses shall be attempted, certain indications must be clearly laid down and contraindications lucidly determined. It should be obvious that all progressive diseases must be excluded, such as syringomyelia, combined degeneration of the cord, multiple sclerosis and Erb's spinal spastic paralysis. It would seem from the requirements laid down by Royle and Hunter that only those cases are suitable which permit of the demonstration of what is termed plastic tone, by means of lengthening and shortening reactions and "hung-up" reflexes. This means that in cases in which increased spinal activity, contractures and fibroses mask plastic tone, the patients are unsuitable for operation. This is not because of the absence of plastic tone but because the other changes are such that they do not permit of any successful reeducation following what seems to be a minute and temporary change in tone.

CONCLUSIONS

1. Histologic evidence points to the dual innervation of skeletal muscle from the cerebrospinal and sympathic nervous systems.

2. Experimental removal of the sympathetic trunks in cats produces no effect on normal tone which can be observed or recorded. Evidence in the literature is equally divided upon this point.

3. The onset and maintenance of decerbrate rigidity in cats is unchanged after the removal of the sympathetic innervation to an extremity. With the exception of Royle's work on goats, the evidence in the literature is in agreement on this point.

4. The problem of muscle tone is extremely complicated, and one or all of several mechanisms may be responsible for changes in muscle tone.

5. There is at present no accurate clinical method for measuring changes in muscle tone.

6. Kymographic tracings of tendon reflexes, faradic stimulation, active and passive motions and tremors before and after removal of the sympathetic nerve supply have shown no change in cases of paralysis agitans, postencephalitic Parkinson's disease or lateral sclerosis.

7. Patients with progressive or systemic degenerations of the spinal cord are unsuitable for the operation of sympathetic ramisectomy.

8. Patients in whom increased spinal activity, contractures and fibroses mask plastic tone are unsuitable for operation.

9. Lengthening and shortening reactions and "hung-up" reflexes alone are insufficient indications for operation.

10. Three patients with Little's disease and one patient with a traumatic lesion of the spinal cord have been operated on too recently to draw final conclusions as to the results obtained.

11. One patient with cerebral hemiplegia has been improved following operation, although no graphic record of this improvement can be shown.

12. The sympathetic nervous system may have some function dealing with the metabolism of muscle so that under certain conditions the contractility of a muscle may be changed by the removal of sympathetic impulses. Such a function would probably be chemical in nature.

SPECIAL ARTICLE

CLINICAL AND ANATOMICAL CONTRIBUTIONS ON BRAIN PATHOLOGY

S. E. HENSCHEN

Formerly Ord. Professor af Innere Medicin, Karolinska Institutet

STOCKHOLM

(ABSTRACTS AND COMMENTS BY WALTER F. SCHALLER, M. D., SAN FRANCISCO)

FIFTH PART: APHASIA, AMUSIA AND AKALKULIA

Henschen relates that on Feb. 28, 1912, the date of his sixty-fifth birthday, he was obliged by statute of age limitation to retire as director of the medical clinic of Stockholm, and thereby from his usual clinical and laboratory activities. This outlook was not promising for original work in brain research, as future investigations depended on a review of personal, clinical and anatomic material and of the subject literature. However, undismayed and after different publications on disease of the heart (1913), the auditory center (1918), the center for smell, and the visual center (1917), Henschen reverted to the great aphasia problem which had interested him for over a period of thirty years.

The problem of speech depends on a correct grasp of function, form and organization of brain sense areas and their centripetal and centrifugal connections, and especially of the centers of sight and hearing. The knowledge thus obtained will serve as fundamentals for further psychic investigation, which must not rest on purely fantastic conceptions, but must have a firm clinico-anatomic basis.

The plan adopted in 1890 is followed in this work, namely, systematic assemblage of case material and a critical analysis of selected cases, with only such deductions as are warranted by the facts. In all, 1337 cases are individually abstracted from the author's experience and from the literature. There are convenient tables in considerable numbers and in accessible form, although many have been cut down from the originals because of typographical and economical reasons. The work is richly illustrated by original anatomic photographs, cuts and selected reproductions.

The work is in three large quarto volumes. The first volume deals with aphasia, amusia and akalkulia; the second, with sensory aphasia; and the third, with motor aphasia and agraphia.

Aphasia.—Reports on the first group of aphasia cases, Cases 1 to 29, were previously published by Henschen, but he includes these cases also in this treatise. They include a variety of clinical forms and of localization of lesions. A case of particular interest, No. 3 (Clara Nilsson), presented a bilateral temporal lesion which was accompanied by a persisting word deafness, although reading and writing were unaffected. Motor speech was complicated only exceptionally by paraphasia and amnestic aphasia. Wernicke and Liepmann have insisted that word deafness in left-sided temporal lesions is only a transitory symptom, and this case would confirm this opinion in a negative sense. It is generally accepted that expressive speech is dependent on comprehension of the spoken word. This was not true in this case. The psychic word perception (Wortsinnverständnis) was present but not the auditory word perception (Wortlautverständnis).

The independence of reading on auditory word comprehension in this case, as in other reported cases (Quensel), would take support away from Wernicke's teachings on aphasia. According to Henschen, there are independent cortical centers for comprehension of the spoken word and for reading. Further, this case shows that there are two auditory word centers: one for auditory word perception and another for psychic word perception. It is manifest early in the work that Henschen is a strong proponent of the notion of definite cortical centers for various speech functions.

Two cases of left-sided temporal tumors of great extent showed no trace of word deafness, but amnestic aphasia and paraphasia were marked. Because

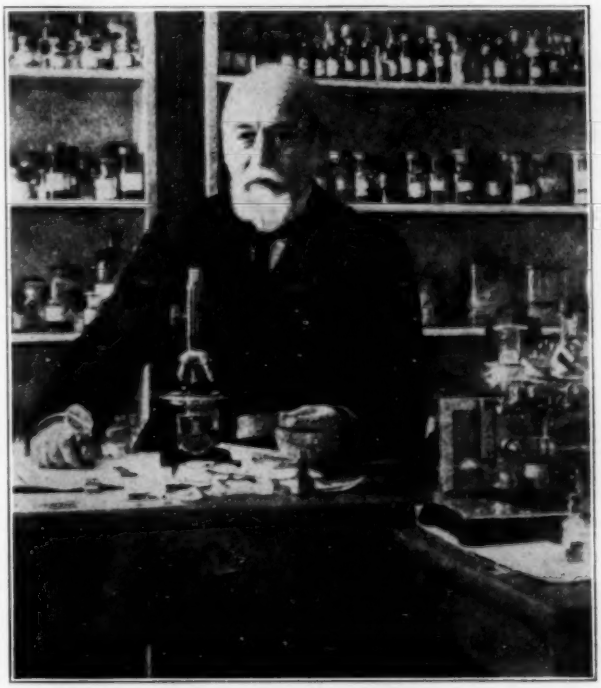

Fig. 1.—Dr. Henschen, 70 years old, on Feb. 28, 1917.

of the slow growth of brain tumors, a vicarious action of the right temporal lobe must be considered. In two cases (Cases 4 and 5) of left-sided temporal abscess, word deafness was absent. Word deafness in left-sided otitic temporal lesions should not, therefore, be contraindications for operation.

An interesting case (Case 6) of transitory alexia due to hemorrhage involving a portion of the angular gyrus is reported in the case of a highly cultured professor of surgery, who occupied his vacation time by diligent study of literature. He would spend the entire night reading, Horatius by choice. Suddenly stricken unconscious, with epileptic attacks, he eventually recovered completely, and was able to resume his professional duties. Word blindness in this case was not accompanied by word deafness, but by a word amnesia which persisted for a considerable period. The patient finally succumbed in another apoplectic attack. At the necropsy, in addition to the fresh hemorrhage, two

old hemorrhages were found, one in the cerebellum and one in the angular gyrus. The genesis of the latter lesion is explained by increased local blood pressure in a region of local psychic effort causing a rupture of a previously diseased vessel.

Liljenberg (Case 8) is quoted in support of the contention that visual word impressions in reading are independent of auditory word impressions through

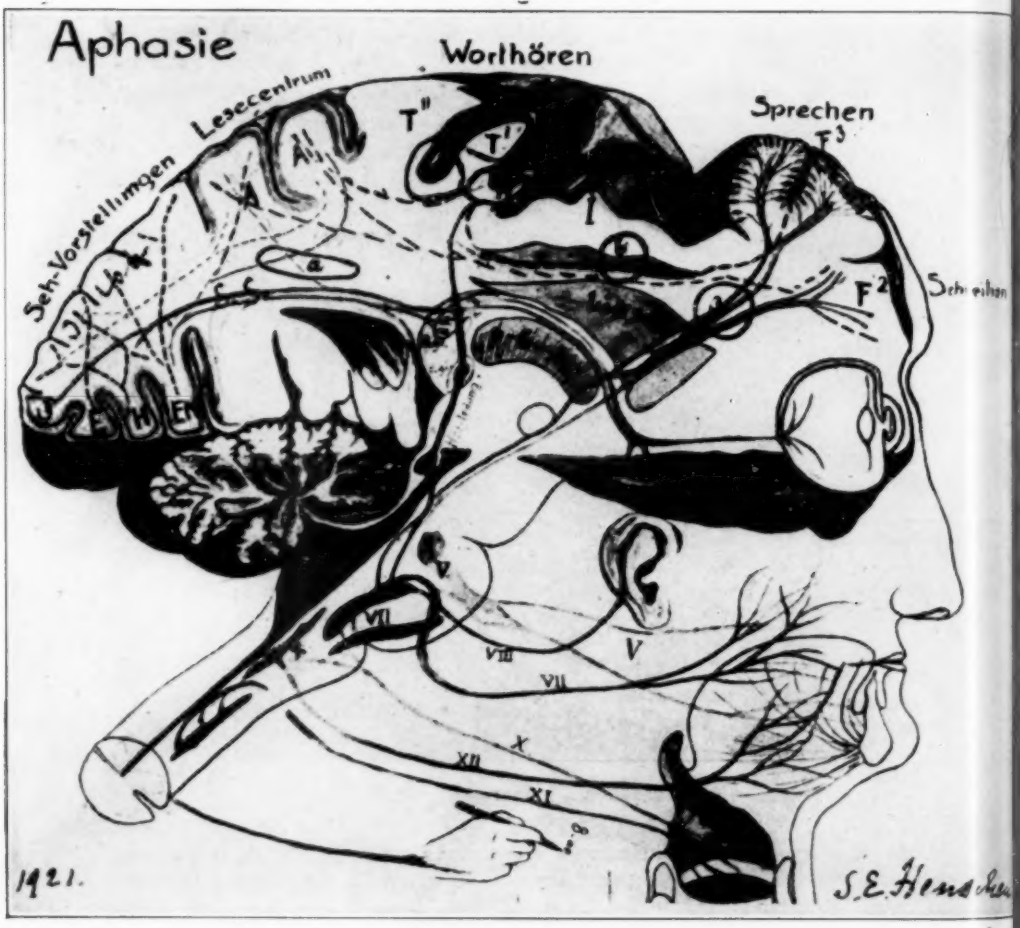

Fig. 2.—Scheme of aphasia, according to Henschen.

hearing, and therefore contradicts the theories of Wernicke and Déjerine. An educated woman lost the power of reading and writing. Only slight motor aphasia was present. No trace of word deafness was present, although there was slight paraphasia and amnestic aphasia. Anatomically, a large area of softening was found in P_2 and A, and independent of this area a large additional area in the occipito-temporal convolution. This case indicates a sharply defined center for reading.

Five cases of a combined lesion in the temporal lobe and angular gyrus are analyzed. Whereas in four cases word blindness was present, in two cases word deafness was questionable. This fact points to the greater susceptibility of the visual center. In all cases, amnestic aphasia and paraphasia were present.

In the group of motor aphasia, four are recorded with lesions in F_3, one bilateral. One case (right-handed) showed anatomically the classical lesion of the foot of the left third frontal convolution without motor aphasia. Attention had already been called to the case by Niessl v. Mayendorf in this

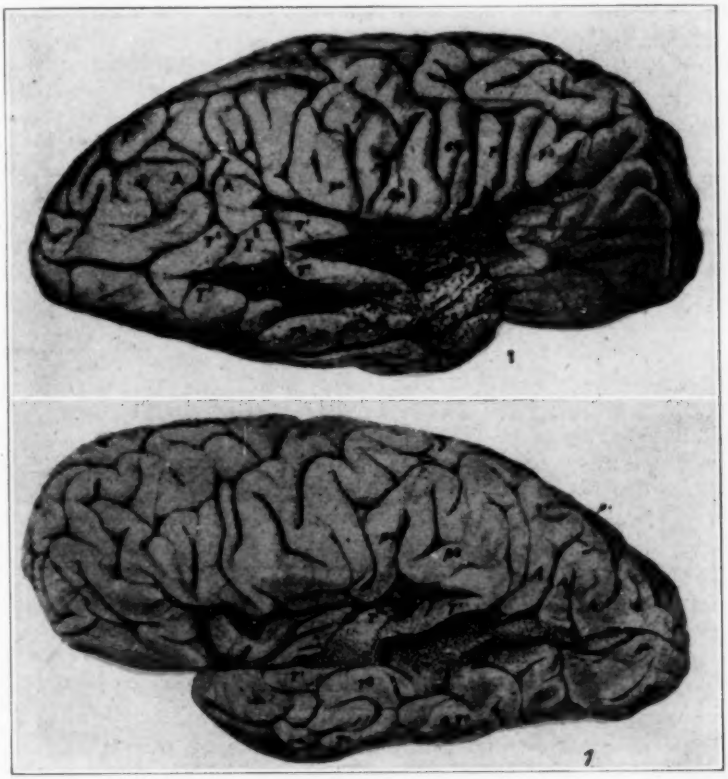

Fig. 3.—Henschen's Case 3; persisting word deafness due to bilateral temporal lesions.

connection. Although the patient was able to speak directly after the attack, the possibility of a vicarious function of the right hemisphere is mentioned, which might have been established some time before in unrecorded periods of speech defect.

Two cases of sensory-motor aphasia are recorded in which combined lesions of the foot of the second frontal convolution and of the angular gyrus in the left hemisphere were followed by alexia and by agraphia. The most significant of these cases from the standpoint of the anatomic lesions is Case 22. While writing a letter, the patient was obliged to discontinue because she could no longer use the hand in writing. One hour later, having

gone to bed in the meanwhile, she could not read what she had written. This case is held as further evidence of lesions (thrombosis) occurring in consequence of local psychic functioning.

Amusia.—Concerning amusia, an excellent historical review precedes the critical analysis of the case abstracts of which there are 251.

Rhythm is the principal constituent factor in music; the second essential factor is pitch. In addition to these basic requirements, melody, modulation, intonation and accentuation are required to produce music. Their disturbance is defined as amusia. Amusia promises to have a greater form variety than aphasia. Rhythm is a kinesthetic sense, and is seldom lost in comparison with pitch and melody. Being a basic requirement of music, it is suggested that dancing may further musical education. The localization of rhythm may be sought near the muscle sense in the parietal lobe. Knoblauchschen's classification of amusia is accepted.

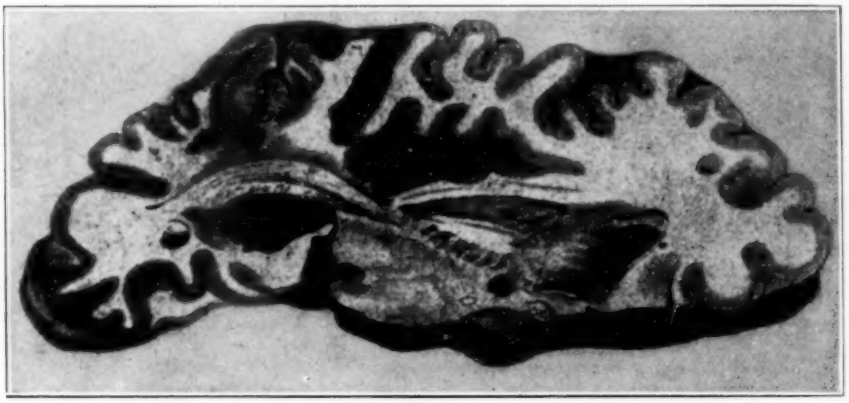

Fig. 4.—Henschen's Case 6; transitory alexia due to hemorrhage involving the angular gyrus.

A. Motor.—(*a*) Vocal music with or without words; (*b*) note agraphia; (*c*) instrumental.

B. Sensory.—(*a*) Music deafness with para-amusia and amnestic amusia; (*b*) note blindness.

Wortzen relates Case 365, that of a ballet dancer and excellent musician, aged 48, who was stricken with apoplexy and suffered in consequence a temporary left-sided hemiplegia. Although his general mentality and understanding were scarcely affected, comprehension and expression of music were strikingly affected; his rhythmic sense had entirely disappeared. There was some dysarthria. When he attempted to beat a measure or to keep time in dancing, he failed utterly. He had no insight into these mistakes. Nothwithstanding, he greatly enjoyed listening to music. Energetic attempts to relearn instrumental music met with failure. He was unable to recognize false notes when he played. In this case, rhythmic sense was affected as well as judgment of pitch.

Forster tested out the musical ability in a patient (Case 135) who became aphasic after a stroke. The analysis showed that the sense of pitch was well preserved, whereas rhythm was faulty. Chopin's Funeral March was classed

as a song, and an Andante from Mozart's C Major Sonata was regarded as a waltz. A number of examples of the frequent occurrence of preserved rhythmic sense and loss of the sense of pitch are detailed.

Disturbance of the higher musical faculties may be detected by a musical clinician in the lack of proper evaluation and recognition of chords, tone colors and intonation. Henschen gives a classic description of these variations, which impresses the reviewer that the author must possess a musical understanding far above the average.

Musical memories have been completely lost. Two striking examples are recorded in professional artists, whose cases are quoted as evidence that musical ability in its comprehensive state is a psychic entity and is entirely independent of aphasia. It is striking that an incitement even of mild degree can often overcome musical amnesia. This fact would point to an automatic mechanism of song.

Concerning the relationship of aphasia and amusia, three groups are segregated:

GROUP 1. Aphasia without amusia. There are eighty-nine cases in this group.

GROUP 2. Aphasia with amusia. This group comprises forty-three cases, that is, about half the number of the foregoing group.

GROUP 3. Amusia without aphasia. There are only sixteen cases in this group.

These cases of isolated amusia, however, throw a clear light on the specific nature of, and the separate centers for, music. The fact that in children the music sense is developed before sensory speech, and that musically gifted children can sing before they can speak, points also to a song center apart from a speech center. Further, singing consists of a simpler mechanism than speech in that the vocal cords and breathing only are necessary for singing. Nevertheless, because of the frequent association of aphasia and amusia, the two centers are conceded to be near each other. The author concludes, after an exhaustive discussion of the subject, that the center for vocalization lies in the lower border of the pars triangularis of the third left frontal convolution and represents, analogous to Broca's area, a higher psychic center. A considerable number of persons with motor aphasia (aphemia) may sing a vocal text.

The localization of the receptive center of auditory music as compared with the center of auditory speech lies in the left temporal pole. These conclusions are based on nine positive cases in which a lesion in this locality was associated with amusia, and in twenty negative cases in which there was no amusia, with an intact T. pole.

Note blindness and word blindness are not necessarily associated. The center for the former points toward the vicinity of the angular gyrus (cases of Déjerine, Marie-Sainton, Redlich) or the intraparietal fissure (cases Touche, Galliard, and Bernard). The center is still hypothetical.

Amusia instrumentalis: Henschen proposes for this deficiency the term "music apraxia," or instrumental amusia. Only thirty-eight cases are recorded of the ability of persons with aphasia to play musical instruments; of these, twenty-one had amusia instrumentalis, but seventeen, notwithstanding some kind of amusia, had not lost the power of instrumental (motor) music. Either the center for the sense of tone or the motor system is involved in music apraxia. Anatomic data are in accord that there is an instrumental motor

center in the foot of the second frontal convolution or in its close vicinity. There appears to be a special center for violin players.

Akalkulia.—The arithmetical sense is of great practical significance for the intercourse of every-day life, for business and commerce. The arithmetical knowledge of a child of 5 or 6 years is the inheritance of the accumulated progress in this endeavor of centuries. Native and uneducated people have the most primitive means of expressing the simplest numerations. The Pitta Pitta stone folk of Queensland designate 1, roo; 2, pa-koo-la. In reckoning, parallel lines are traced in the sand, and when the numeration is complete he calls pa-koo-la for every two of them. It is of considerable interest that after an aphasic attack an educated person will revert to simple means of expression similar to those of these stone folk. They resort to parallel lines, count fingers, or repeat a given word, for example, five times to designate five.

From the fact that stones were used in the beginning for reckoning, Henschen has suggested the term "kalkulia" and "akalkulia" to designate the ability and the loss of ability, respectively, to calculate. These terms and functions are analogous with similar terms and functions of speech and music. "Kalkulia" and "akalkulia" are used in both general and specific terminology. For the special form, "cipher deafness," "cipher aphemia," "cipher blindness" and "cipher agraphia" are used. In a general sense, akalkulia is the inability to form combinations of numbers. Leading mathematicians have been questioned as to their opinion as to the physiologic process of calculation. There appears to be no unanimity as to whether the process is predominantly optic or acoustic. In children the visual sense serves in their early learning, but later addition and multiplications are automatically performed. The multiplication tables are inwardly heard, and in this wise the auditory sense and auditory memories are called into use. The skilled mathematician reckons with optic combinations analogous with the Chinese hieroglyphics. The material available consists of 260 observations. No observation is available in a disturbance of higher mathematical ability, and most of the reported cases deal simply with inability to add, subtract or multiply.

In the literature, the masterly work of Moutier on aphasia, and the special investigation of Peritz[1] on traumatic war cases, are especially mentioned. Exact observations in a sufficient number of cases regarding the parallelism of word deafness and cipher deafness are lacking to warrant any absolute deductions as to localization, but it is certain that auditory word and cipher perceptions have different centers.

Regarding the motor speech and cipher mechanism, the observations are more numerous. An analysis of tables and of anatomic data leads to the following deductions: 1. Cipher aphemia and word aphemia do not run parallel, although they often occur together. 2. Cipher aphemia is not influenced by word deafness. 3. An isolated cipher aphemia has not occurred in the material

(1) Concerning other speech elements, four cases only of isolated cipher blindness were found (not accompanied by word blindness).

(2) In 122 cases of word blindness, seventy-one persons could read ciphers, which speaks strongly for separate centers.

(3) Fifty-one persons had both word and cipher blindness, which speaks for the proximity of these centers. Cipher blindness and akalkulia are associated in the majority of cases. This fact proves that the ability to read figures (optic or mental) has a great significance for calculation.

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1. Peritz: Deutsch. Ztschr. f. Nervenh. **61**:234-340, 1918.

(4) Cipher agraphia and word agraphia do not run parallel, for in more than half the cases of word agraphia, cipher agraphia does not occur.

An effort was made to determine how far the aphasic elements entered in akalkulia. Whereas word deafness, word blindness, word aphemia or word agraphia influenced akalkulia slightly, inability to copy (akopia) often was accompanied by it.

Touche (Case 337) reported a case in which the patient was unable to articulate, read or write a cipher. Nevertheless, he could calculate; he was able to do small sums correctly.

Five cases of akalkulia are reported in the literature—Schuster (310), Righetti (305), Gossen (148), Moutier (235 and 219), in which the three cipher elements were conserved. Henschen interprets Touche's case as one in which the patient calculates with the acoustic sense. The other five cases in which

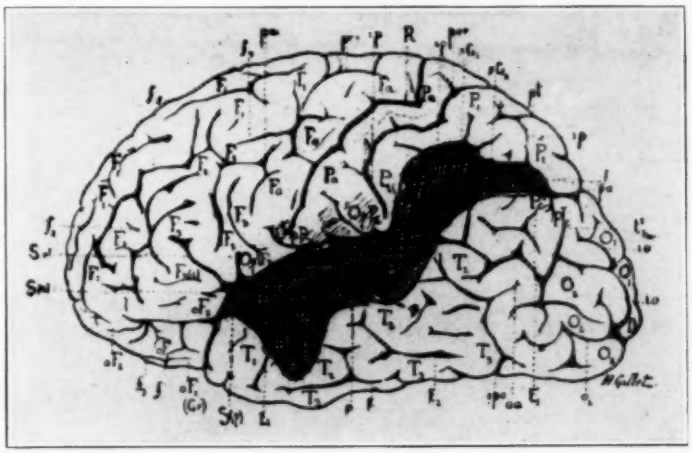

Fig. 5.—Touche's Case 337; cipher aphemia and cipher agraphia with preserved ability to calculate.

there was akalkulia are incomplete in analytic details as to the extent and character of ability to calculate.

All data considered, the author concludes that there exists in the brain an independent system subserving arithmetical processes and that it is independent, or nearly so, of the systems for speech and music.

Anatomic Considerations: Localization: Extensive lesions of the left hemisphere occasion not only total aphasia, but also cipher blindness, cipher agraphia, and often akalkulia, but infrequently cipher aphemia. The ability to count when the motor speech center is involved points toward the right hemisphere as taking over this automatic function.

Cipher reading has its localization in the dorsal bend of I_p in the ventral adjacent part of P^1. Lesions of the gyrus angularis are often accompanied by cipher blindness, and this is total when the region referred to above is implicated. It is suggested because the region of cipher reading is supplied by the anterior cerebral artery, it is less often affected than the region of word reading supplied by the more used middle cerebral artery.

The localization of cipher writing also appears to be identified with the region between A and P^1. As the motor components of writing are carried

out through a motor center, the foot of F^2 is indicated but not definitely established.

The concluding chapter of this division deals with the various theories of akalkulia and especially the various explanations offered to explain the fact that alexia is frequently unaccompanied by cipher blindness. Schuster's theory based on the interrupted association of optic word pictures and sound pictures producing alexia but sparing other association paths, would permit enumeration without true imagery. This theory is particularly unacceptable to the author, because he is a strong exponent of the independence of the optic word picture on auditory memories.

SIXTH PART: SENSORY APHASIA

Temporal Aphasia.—The literature on aphasia has attained a considerable volume: almost 3,000 articles. Notwithstanding this large amount of material, satisfactory progress has not been made in our knowledge of the subject. This is in part due to a lack of unanimity as to the proper method of aphasia research, clinico-anatomic or philosophic—psychologic; to the dominating influence of the doctrines of Wernicke and Lichtheim hampering original research; and to the number of poor clinical case reports and the still poorer anatomic reports, many of which show a contradiction in the text description and in the anatomic reproductions. A number of brilliant exceptions are found, however, in the works of Fouche, Moutier and Bernheim, and of different German investigators. An analysis of suitable and available reported cases is necessary to pioneer work, to which the author has devoted his labors.

Other inhibitory movements have been constructive schematizations and their elaboration from purely philosophic and psychologic considerations; and the lack of knowledge of the location, borders and organization of the special sense centers, especially the auditory and visual centers at the time of the birth of the aphasia theory.

Whereas Wernicke, on the basis of Meynert's anatomic studies, placed the auditory word center in T_1, and thereby founded his theory of aphasia, he considered that this area was not identical with the entire cortical distribution of the acoustic nerve, but only a part of it. Henschen ("Ueber die Hörsphäre") has localized the center for general audition in Heschl's convolution. An historical review, starting with an observation of Ambrose Pare in 1538, is given concerning this subject, and among others who have made contributions are mentioned Ferrier (1875), Flescig, Mills (1891).

A tabular analysis of cases leads to the conclusion that a bilateral lesion of Heschl's convolution and T_1, T_2 and T_3, produces absolute and persisting deafness, whereas a bilateral lesion of T_1, T_2 and T_3 does not produce deafness, providing Heschl's convolution is spared. Anatomic considerations of Heschl's convolution deal with location, cyto-architectonic, and myelinization time.

Temporal aphasia is dominated by word deafness. An inquiry into the location and character of word deafness is therefore pertinent. The term "word deafness" is credited to Kussmaul (1879). As early as 1869, Bastian differentiated the two types of sensory aphasia—the acoustic and the optic. Bastian is therefore credited by Henschen as the founder of sensory aphasia. Bastian credits Wernicke, however, with the merit of having determined the region of the brain at fault in word deafness, but believed he was far from correct in assuming that alexia and agraphia were necessarily consequences. A review of the views of later contributors (Monakow, Déjerine, Mingazzini

and others) seems to point to a confusion as to whether there is one kind of sensory aphasia or two, and further as to the identity of general audition and word audition. As word deafness includes impaired function of letters, syllables and sentences, as well as of words, Henschen proposes the term "speech deafness" instead of word deafness.

The comprehension of syllables and short every-day words is localized in T_1, whereas the psychic comprehension of coordinated word sounds lies in other parts of the temporal cortex and with the help of internal association fibers permits contacts with tactile, optic and gustatory concepts. If this conception is correct, then word sounds pass through a series of stations before they are properly "sense words." In any event, there are three separate localizations possible in the temporal lobe: (1) Heschl's convolution, (2) posterior two thirds of the first temporal convolution, and (3) the remainder of the temporal lobe (cortex and medulla)—an extensive station.

Internal Speech: This term ("das innere Wort") is said to have originated with William von Humboldt. He defined this as the content and processes of consciousness, which are expressed by speech. It is a theme that has interested philosphers and psychologists as well as brain physiologists. Thought and speech are not identical, as they may exist separately, and much confusion has arisen out of disregard for this factor. The author agrees with Hughlings Jackson that "internal speech is not necessary to perfect logical thought." Henschen postulates two cardinal and inter-related elements in internal language, namely, internal sensory speech, which is word wealth serving as psychic symbols, and internal motor speech, which gives the former a more definite form.

Concerning the localization of word deafness, eight tables of cases are analyzed to elucidate this problem: The fourth table deals with unilateral diffuse lesions of the left temporal lobe; Tables 5 and 6 deal with lesions of T_3, $T_2 + T_3$, and T_2; Table 7 with lesions of $T_1 + T_2$; Tables 8 and 9 with lesions limited to T_1; Tables 10 and 11 with bilateral temporal lesions with and without disturbance of internal speech, and Table 12 with jargonaphasia with unilateral lesions.

On page 75 of this volume occurs an exhaustive discussion of the anatomy, both gross and histologic, of Heschl's convolution.

We have up to the present time no absolute proof that a cortical lesion limited to the posterior third of the first temporal convolution occasions speech deafness. Nevertheless, there are a great number of negative cases and also positive cases which permit little doubt that lesions of the middle and posterior portion of T_3 are accompanied by speech deafness. Purely cortical lesions limited to a small area of the brain cortex are infrequent, and in the speech area they are usually combinations of the different temporal, angular gyrus, and parietal lesions. A number of different combinations of these groupings arranged in tabular form are discussed, consisting of lesions of the posterior third of the first temporal convolution and adjacent areas: $+ A$; $+ P_2$; $+ A$ and P_2.

It appears that lesions of the posterior part of T_1 are characterized by sentence agrammatism, whereas defects of the middle portion of this lobe produce word agrammatism. The deduction may be drawn that the word sounds (syllables) are registered in the middle portion of the lobe, and then arranged in sentences in the posterior portion, which is a higher center; supporting this idea of a primitive function of the middle portion is the anatomic fact (O. and C. Vogt) that it is earlier and more extensively myelinized than the posterior portion.

The acoustic perception of words and the storing up of word memories are two different psychic processes and are served by two different brain centers. The bilateral destruction of T_1 does not interfere with normal speech (motor). Cases 3 and 373 are quoted in support of this contention. They are both cases of preserved motor speech with practically complete speech deafness in bilateral lesions; neither was alexia present in these cases.

Word blindness is discussed in relation to word deafness. Word blindness was found lacking in thirty-five cases of temporal lesion in which there were twenty cases of word deafness. In consequence of this fact, Wernicke's theory that word blindness is a direct consequence of word deafness and that reading is transmitted through T_1 is definitely refuted; and a considerable portion of the literature on aphasia has been refuted, as the authors based their conclusions on this faulty premise.

The Wernicke school includes agraphia as a constant and necessary consequence of word deafness. In twenty-six cases of agraphia out of a total of thirty-three, word blindness explained the agraphia without recourse to an explanation by the presence of word deafness, and in two cases word blindness was absent.

Word deafness, although frequently accompanied by akopia, does not in itself condition it.

Although an isolated lesion in T_1 does not cause aphemia, yet there is much contradiction in published reports concerning the presence of aphemia (excluding paraphasia) in extensive and diffuse lesions of the temporal lobe (page 87). It is concluded, however, that word deafness in itself does not cause aphemia.

Amnestic aphasia may be due to a physiologic cause such as senility and fatigue; it may be due to nervousness, psychosis or intoxication; or it may be due to focal lesions. It is a symptom that may be due to angular gyrus and frontal lobe lesions, as well as temporal lesions, which are here considered. "Amnestica verborm" is the nucleus of aphasia. Word memory is not an elementary psychic function, but a complicated one depending on acoustic, optic, tactile, gustatory and olfactory elements. The analyses show that lesions of T_2 and T_3 are particularly liable to be accompanied by amnesia, and also that word deafness does not necessarily imply amnesia. Otitic abscesses by their extension through the tegmen tympani invade the lower part of the temporal lobe, and it appears to be a clinical fact that amnestic aphasia is one of the earliest symptoms.

We have insufficient data to determine whether lesions of the right temporal lobe occasion amnestic aphasia. Edinger has reported a case of extirpation of the whole right temporal lobe without any effect on speech, and Henschen a case of multiple softenings in the right temporal lobe with ultimate complete speech restoration.

Paraphasia must not be confused with jargonaphasia. The classification of Wyllie defining a motor and an acoustic form is not acceptable to Henschen. As motor speech is not lost in bilateral destruction of T_1, the lesion of paraphasia may be sought for in T_2 or T_3, and even slight lesions as in otitic temporal abscesses may cause it. In twenty-five cases of abscess, paraphasia was noted, and in only ten of these word deafness was present.

Echolalia, a forced reflex or meaningless mechanical repetition of words, is a function of the right temporal lobe (T_1); the conscious repetition of words, of the left.

Function of Right Temporal Lobe: The function of the right temporal lobe is a much discussed problem. Whereas Lichtheim and also Neissl von Mayendorf consider word deafness only a transitory phenomenon, other authorities, such as Mirallié, assert that it is more or less permanent. In order to elucidate the problem of the prognosis and duration of word deafness, 800 cases of temporal lesions have been reviewed. Of these 104 (13 per cent.) only are sufficiently complete to make the following deductions:

1. The prevalent notion that the right first temporal convolution can completely replace the function of the left lacks scientific confirmation.

2. The recovery or substitution is only partial in about 60 per cent. of the cases.

3. Complete and permanent word deafness persists in about 30 per cent. of all cases.

The length of time for alleged complete recovery after a stroke of paralysis is given variously from a few weeks to two or more years. The time required for partial recoveries have ranged from two and one-fourth to eight months, and even years. The persisting cases have been observed for from four to five years, and after this period must be considered as incurable.

Transcortical Sensory Aphasia.—Lichtheim's transcortical aphasia, according to the theory of its author, is due to the interruption between the sensory and the motor centers on the one side, and the theoretical concept centers on the other. In consequence, comprehension of the spoken word is lost with its sequelae in the one instance, and lack of spontaneous impulse of motor speech on the other, while repetition of the spoken word without understanding is preserved. Whereas transcortical sensory aphasia has a definite clinical syndrome, two different anatomic groups are recognized—one, a general brain atrophy with particular involvement of the temporal and frontal lobes; the other, due to a focal infection in the temporal cortex or subcortical white matter. In an analysis of four groups, the principal lesions are always found in the speech centers; therefore the term "transcortical" is inexact. In all cases, moreover, there is only partial speech deafness, with preserved word sound (Wortlautgehör) but with loss of word meaning (Wortsinnes). The common lesion in a number of complicated anatomic studies is a partial lesion of T_1. The concept center or paths are involved, but the receptive word sound center and its connection with the word meaning center is intact or nearly so.

Otitic Temporal Abscess.—A separate chapter is devoted to a discussion of this subject. Amnestic aphasia and paraphasia are the characteristic symptoms of a lesion that generally begins in T_3. When there is a definite word deafness, an extension of the lesion to T_1 must be considered. Extradural abscesses may, however, occasion marked aphasia. Motor aphasia has been described in about half the cases reported. It is, however, much to be doubted that a definite or marked aphemia exists in temporal abscess. Undoubted cases of extensive abscess formation occur in T_2 without the slightest aphasia. This is to be explained by the encapsulation of a small focus beginning in T_3, which displaces rather than destroys brain tissue as it increases in size.

Angular Gyrus Aphasia—Pure angular gyrus lesions are rare. In the entire literature only five have been found in the left hemisphere, and one in the right hemisphere, but the size and location of these lesions in combination with the clinical symptoms have shown such conformity that they suffice to put the physiologic-psychic function of the gyrus on a firm basis. In addition

to these cases, there are nine others in which additional lesions occur but which are without signification for alexia. Complicated cases with A lesions are not rare, about 250 being recorded. The characteristic symptom is word blindness. As an introduction to the discussion of the subject, the different methods of learning to read are recalled, of which there are three: that of the combination of letters read aloud to form syllables, then words; the phonetic method, which dispenses with the letters but utilizes syllables and words in conjunction with the pronunciation of the same; and the writing-reading method, in which the pupil by writing, learns to read. By the first two methods, optic education is by means of the acoustic center; by the writing-reading method, the optic center associates with the kinesthetic as well as the acoustic center. The deaf and dumb can utilize only the optic and kinesthetic centers. On the degree of use and practice of the optic center in reading depends the dependence or independence of this center on the other associated centers. For instance, the independence of the optic center on the auditory center develops particularly in readers and scholars who by book reading seldom call the acoustic center into play. The scholars who learn a new language, such as hieroglyphics or the Assyrian cuneiform script, can read these with understanding before the pronunciation has been determined.

The analysis of cases of word blindness (403 cases) show great diversity of form. Nineteen groups are segregated; certain groups have particular interest—Group 5 consists of the ability to recognize correctly letters but not words. These two operations are therefore considered distinct and have different cerebral locations. Under pathologic conditions, the higher functions may be lost first. In Group 8, the reverse holds: words of even three or four syllables can be read, but not a single letter. The word form in this instance is likened to a picture, a seal or a decoration—these pictures are often recognized without meaning, but in some cases with understanding dependent on preserved associations.

Anatomically, the angular gyrus is described as the posterior part of the inferior parietal lobe and separated sharply from the superior parietal lobe by the intraparietal fissure; toward the temporal lobe, its boundary is not sharply defined, but merges into T_1 and T_2. Cyto-architectonic studies of this gyrus show that this area is sharply defined from its neighboring structures (Elliot Smith, Brodmann).

Reading without understanding (sinnlos Lesen), Table 23, may be due to several causes: lesions of the angular gyrus, general brain atrophy, or atrophy predominating in the frontal and temporal lobes, and cases without angular lesions but in such close proximity that the function of this convolution is affected; or due to disturbances at a distance (Fernsymptom). Analysis would indicate that the right hemisphere can serve only a mechanical function of reading and cannot subserve the higher psychic function of reading.

In all the pure cases of alexia, agraphia when tested in its several forms, was present: loss of spontaneous writing, dictated writing and copy. Word comprehension and expressive speech and repeated speech were not found disturbed. The absence of word deafness is of fundamental importance in the conception of alexia. Amnestic aphasia and paraphasia were, however, frequent accompaniments. Word blindness is as a rule accompanied by right-sided homonymous hemianopsia. They have nothing in common in function, but on account of the anatomic relationship are commonly found together. When the lesion is entirely cortical, word blindness exists without hemianopsia.

Mind blindness is a separate phenomenon from word blindness, and practically without exception was not found complicating lesions of the angular gyrus. Word blindness is a defect in recognition of acquired writing symbols; mind blindness is a failure of recognition of objects of any description learned by previous experience.

Henschen offers an hypothesis of the physiologic function of the angular gyrus. It is a transformer station where the optic word picture having a definite energy content is transformed into terms of other associated special sense values, forming a complete psychic concept.

Occipital Aphasia.—Twenty-nine cases of occipital lesions are tabulated and analyzed. Six of these are bilateral lesions; twenty-two, left-sided lesions; and one, a right-sided lesion. Word blindness, agraphia and mind blindness frequently occur together in occipital lesions, but are not necessarily or uniformly associated. Why in certain cases only mind blindness occurs, in others only agraphia, and in some mind blindness and agraphia, is unexplained except for the hypothesis that a special writing center occurs in the dorsal part of the occipital lobe. Word blindness occurs in all extensive diffuse lesions of the left occipital cortex. Mind blindness occurs in extensive lesions of the left occipital cortex. Mind blindness occurs in extensive lesions of the lateral cortex O_1 to O_3.

In the discussion of angular gyrus lesions, it was found that word blindness ("Schrift"—writing blindness) was practically never associated with mind blindness. In the occipital lesions, word blindness occurs in this association in more than half the cases. Word blindness may be considered a special form of mind blindness, which is a more elaborate function. There are a variety of gradations from marked mind blindness to slight word blindness, of which optic aphasia is an example. Writers on optic aphasia describe this phenomenon diversely: failure to name an object by sight, but ability to name it by palpation; or, the script may be recognized but not named. Cases are reported by Peters, Vorster, Hosch, Liepmann, Müller, Lissauer, Jack, Nodet. In the cases of Müller and Lissauer, a touch agnosia was present.

Combined Lesions of the Temporal, Parietal and Occipital Lobes and Angular Gyrus.—The author gives eleven different combinations of lesions of the temporal, parietal and occipital lobes and angular gyrus, with corresponding tables. Unexplained aphemia is found in certain of these groups. For instance, in combined temporal and parietal lesions, it was present eight times and absent five times. In lesions of T + P + A + O (twenty-five cases), which involve a considerable portion of the whole hemisphere (bilateral as well as unilateral cases), aphemia was present definitely in nine cases as against sixteen (which included the questionable cases). Word deafness in these extensive lesions is generally present; word blindness and agraphia are the rule.

Word blindness plus word deafness plus agraphia, according to the analysis of Henschen, do not necessarily lead to aphemia.

Apraxia is especially mentioned in discussion of T + P + A lesions, in relation to the occurrence of aphemia, with which it may be physiologically associated.

SEVENTH PART: MOTOR APHASIA AND AGRAPHIA

This, the third and largest volume of the aphasia series, contains 319 pages of case reports and text and seventeen pages of reproductions of pathologic specimens. It is dedicated to the memory of the great masters: Bouillaud, Broca and Charcot. After a brief historical account of the subject and general

remarks regarding material, nomenclature and the trend of recent investigations, as well as the inherent difficulties of aphasia research, important chapters are devoted to: speech and the frontal lobes; word blindness and word deafness in aphemia; boundaries of Broca's area; progression and regression of speech defects from frontal lesions; analysis of insular and central ganglion lesions; agraphia; the rôle of the right hemisphere and a general discussion and criticism of the prevalent notions and doctrines of motor aphasia. Marie-Moutier's nomenclature and especially the term "anarthrie" as applied to motor aphasia is unacceptable. It is pointed out that Leyden (1867) first used this term as synonymous with bulbar speech defects, and later it was used by various writers as synonymous with subcortical motor aphasia. According to the proposers of the term "anarthrie," it is not pseudobulbar palsy or aphasia, and is to be differentiated from the subcortical motor or pure aphasia of Broca unless subcortical be applied to a lesion of the lenticular zone. Henschen, therefore, finds this term and the description of it confusing and contradictory. In fact, the whole conception of Marie to extend Broca's aphasia to include symptoms of Wernicke's aphasia receives sharp criticism from Henschen, who feels that it is a step backward, unscientific and purely constructive. "Anarthrie" should be stricken out of the nomenclature. "Mutism," as a purely clinical conception, is proposed. As regards the genesis of mutism, it may be due to congenital deafness, lack of sensory or cortical innervation, or the involvement of the innervation of the speech musculature (a dysarthritic form).

Aphemia, according to Henschen, is understood as a disturbed motor coordination of word construction due to a lesion in the foot of the third frontal convolution—a higher psychic center—to which sensory speech impulses arrive and are coordinated. Anarthria or dysarthria is a purely functional motor disturbance, due to a defect in the tongue, lip and larynx mechanism.

Determination of right-handedness or left-handedness is often either overlooked or not noted in clinical reports. Statistics show that about 10 per cent. of all people are left-handed. However, in agreement with Wundt, the author believes that speech is not a function of the hemisphere concerned with manual work, but is a function of this hemisphere concerned with mimicry and gesticulation. Such investigations have not yet been made. The author has been impressed by his own observations in South European countries with the frequency with which the left hand predominates in gesticulation.

Speech and the Frontal Lobes.—Twelve tables of classified frontal lesions are analyzed in connection with motor speech, consisting of isolated and combined lesions of F_1, F_2, F_3 and C_a (ascending frontal).

Most of the cases of F_3 lesions with aphasia are combined with C_a lesions and particularly with the pars opercularis. Five cases of isolated C_a lesions are assembled. A characteristic case of the latter is reported by Onuf (Case 1148). The aphasia came on suddenly—"No aphemia, but dysarthria." The following clinical description fully justifies the distinction: "He spoke in a very thick and blurry manner so that it was difficult for him to pronounce the words distinctly. This, however, was a difficulty he had in moving the lips, tongue and other muscles of articulation. Difficulty in swallowing. No real aphasia. He knew what he was going to say, tried to say, and always succeeded to say, but the words were blurred and indistinct." Necropsy revealed an intact F_3, also the part of C_a which adjoins the precentral sulcus. At the lower part of C_a—C_p was found a blood clot of about a dessertspoonful in quantity. It had almost entirely destroyed the cortical substance of the lower

end of C_a—C_p from the sylvian fissure as high as the level of the fissure that divides F_1 from F_2. Henschen considered this almost unique case as elucidating the function of the inferior C_a, C_p and operculum, as an arthritic, but not phasic (aphasic) localization.

Mingazzini has offered an hypothesis as to the course of motor speech fibers, postulating a separate course of the fibers from C_a (verbomotori), and from F_3 (fascicomotori), in their course toward the neuromuscular speech mechanism. The fascicomotori fibers pass to the putamen, where a new neuron begins in the vicinity of the end of the internal capsule serving the lips and tongue musculature, the lesion of which (lower neuron) produces dysarthria. Henschen's analysis of C_a cases would support this theory, for if fibers from F_3 pass through C_a, aphemia and not dys(an)arthria would result.

Lesions limited to the foot of F_3 are divided into: cortical (seven cases), corticomedullary (twenty-two) and medullary cases (twenty). For the most part, the clinical aspect was one of aphemia or mutismus, but the pictures were complicated, and each case was discussed separately.

Table 7 deals with seventeen contradictory cases. Moutier, it may be recalled, has published cases of F_3 lesions without aphasia, and aphasia with integrity of F_3. Many of the cases, according to Henschen's analysis, are not conclusively contradictory; there are, however, six (seven?) cases that are definitely contradictory and only explained by the eventuality that the patients were unrecognized left-handed persons, or that the right F_3 took over the speech function. However, the latter explanation is not satisfactory in three cases (995, 948 and 709) because of the short term of life after gunshot wounds.

The large number of aphemic cases (eighty-five, including tumors and bilateral lesions) in reported cases of F_3 lesions would outweigh the small number of apparent contradictory cases. It could be explained on the basis of the percentage of left-handed persons.

Neoplasms of the left frontal lobe and of the foot of the left F_3 are tabulated and reviewed. The problem of tumor lesions as compared with vascular lesions is often complicated by pressure and circulation disturbances either locally or generally.

A most important compilation of cases is that comprising bilateral lesions of F_3. A considerable number of these cases have been assembled from the literature (twenty, exclusive of eight of atrophy). There are twenty-four cases in which the foot of F_3 is involved amongst other lesions, and in five cases it alone is involved. The deduction of the author from this study follows: A total destruction of the foot of F_3 bilaterally is followed always by a total aphemia (mutismus). The classical teachings of Broca, Charcot et al. are absolutely verified by the analysis. These cases give no support to the idea of an extended speech area (left pars triangularis, F_2 or C_a, etc.). An atrophy of both F_3 is compatible with preservation of speech even though speech is defective. In none of the twenty cases was the insula or the quadrilateral space (Marie and Neissl von Mayendorf) the seat of any lesion. Only the foot of F_3 determines aphemia.

Word Deafness and Word Blindness in Aphemia.—It must be admitted that the different motor and sensory speech areas (centers) are connected by pathways forming an anatomic system, a lesion of one part of which may affect another and distant part. There is a lack of exact anatomic knowledge of these pathways; at present, we are only familiar with the connection of Wernicke's center in T_1, and Broca's center in F_3 through the fasciculi arcuati

and uncinati. The pathways between the reading center in the gyrus angularis and Wernicke's or Broca's area are imperfectly known or not known.

As congenital deafness is constantly associated with dumbness, especially word dumbness, it is assumed *a priori* that a secondary atrophy exists in F_2. Likewise, it is assumed, but of course not proved, that in the course of time a lesion of Wernicke's center in adults produces secondary degenerations, which explains cases of aphemia in some of the cases of Wernicke's aphasia not otherwise explained by focal processes. Even though knowledge of certain anatomic connections is lacking, the connections between speech centers may be elucidated through symptoms, starting with the established facts that there are different separate and independent speech centers.

When a composite table of frontal lesions and aphemia was examined in relation to the existence of word deafness, 188 cases were negative and twelve were positive in 200 cases. A critical analysis of these twelve cases leaves only one (Case 1026) that is not questionable. The conclusion is naturally reached that even an extensive lesion in F_2 by itself alone cannot produce word deafness, in refutation of the opinion of Wernicke, Déjerine and Monakow.

From the same tables, word blindness was investigated. Of the forty-eight cases examined, nineteen were positive. All these positive cases, however, are questioned on the basis of complicating local or general anatomic lesions, psychoses, intelligence or educational defects. The conclusion is formulated that neither frontal lesions nor aphemia lead to word blindness, although aphemia often leads to alexia, i. e., in the comprehensive meaning of alexia (the loss of articulate expression as well as word comprehension).

Broca's Area.—A chapter is devoted to the discussion of the boundaries of Broca's area. Neither C_a, the pars triangularis or the insular cortex belongs to Broca's area. Lesions of the posterior part of the insular cortex, nevertheless, are associated with dysarthria.

Progression and Regression of Speech Defects.—Chapter 7 discusses the progression and regression of speech defects in frontal lesions. Two lines of investigation are followed: the "phasic" and the "arthritic." Tumors offer the best study of progressive "phasic" cases. When a tumor, for instance, exerts pressure on F_2, speech, although distinct, is slowed. A number of examples are quoted, among which is Starr's case (1255), which showed this condition over a period of two years. Clinicians often overlook this symptom as being the precursor of an aphemia. A limitation of the vocabulary initiates the characteristic symptoms of aphemia. Cases in which tumors have been successfully removed, as in the case reported by Keen (974), with recovery of speech, show a marked slowing of speech in their regressive stage; monosyllables represent the first signs of improvement in regression, corresponding to the inverse order in progression. Progression and regression of arthritic lesions are often similar in type to the phasic lesions.

It may be asserted with definiteness that mutism is the rule with F_3 and combined F_3 and C_a lesions, although no lesion in the lenticular zone or T_1 is present (opposed to Marie). Aphemia is localized in F_3 and dys(an)-arthria in C_a (operculum Rolandi).

Insula and Central Ganglion Lesions.—Over 100 cases of lesions of the insula and central ganglia are recorded. Considerable difficulties of classification and arrangement according to anatomic principle were encountered because of incomplete descriptions and complexity and close relationship of anatomic structures. Interesting is the fifth group of lenticular lesions (eighteen cases), among which four showed no motor speech defect. "The lenticular nucleus plays an

important but not constant rôle in speech. Even a total bilateral lesion may occur without speech defect." (No. 781.)

A survey table of nineteen groups of combined insula, central ganglion tables comprising 124 cases, is found on page 194. The following general conclusions may be drawn from this analysis:

Aphemia, dys(an)arthria, or mutismus, to a certain degree is practically always present in insula-central ganglion lesions. Word deafness and word blindness are not part of the picture. Liepmann's theory that aphemia and agraphia are a special form of apraxia is not confirmed. The speech disturbance is as a rule irreparable, the right hemisphere not compensatory. The analysis of the pure lenticular cases speaks strongly for the localization of the phasic fibers in the anterior, and arthritic fibers in the posterior part of the putamen.

Moutier states: "L'anarthrie est l'impossibilité d'articular les mots en dehors de toute paralysie des mouvements usuels de la langue. Dans le syndrome pseudo-bulbaire il y a paralysie." In an analysis of forty-six cases of speech disturbance which brought up this point, a considerable preponderance of paralysis of the seventh to the twelfth cranial nerves and particularly the seventh and twelfth nerves were noted in dys(an)arthria over aphemia (thirty-one as against seven). Dys(an)arthria is due, as a rule, to a lesion of the inferior C_3 cortex or the descending fibers from this cortical area, which pass through the lenticular zone, and as mentioned above, the posterior part of the putamen. The insistence of Marie-Moutier that there is no connection between dys(an)arthria and paralysis of the speech musculature is wrong. The bilateral innervation of the bulbar nerves, through the vegetative nervous system, often compensates an initial paralysis and may be overlooked.

Chapter 14 deals with frontal lesions complicated with lesions of other speech centers. There are twenty-nine groups of these cases; of especial interest are two groups—one is the combined frontal and insular-central lesions, of which 104 are tabulated in symptomatic and anatomic detail. The aphemia or characteristic symptom in these cases was absolute in only seven cases, and the only plausible explanation was a vicarious action of the right hemisphere. The other group consists of frontal, central-ganglion, temporal, parietal and angular lesions, which include practically the whole speech areas. The findings and symptoms in this group are considered by the author to be a firm support of the idea of spech centers advanced by the older writers and himself.

Agraphia.—Speech and writing as a rule go hand in hand. In a cerebral accident, they are often lost together, and in recovery they both show improvement, although recovery of speech is often more rapid. It is only natural that their functions should be considered in relationship, especially as writing is learned in many instances by spoken word memories.

Wernicke and Déjerine, on the basis of theoretic and schematic considerations, have held that writing is dependent on motor speech and that agraphia in itself is a symptom of motor aphasia. Opposed to this absolute view, Bastian, Charcot, Kussmaul and Bramwell were inclined to admit that there was a center for writing, and Exner localized this on the basis of a small and inconclusive material in the foot of the second frontal convolution. On the basis of an analysis of five cases of localized lesions, four of which were limited to the foot of F_2, Henschen feels justified in placing in the foot of the second frontal convolution a center for writing analogous to Broca's center for motor speech.

Writing is a complicated process. This process consists of optic, reading practik, acoustic and motor components. These are of occipital, parietal, temporal and frontal origin. If a link in this chain is broken, agraphia ensues. The special function which lies in F_2 is a higher psychic center, a concept center for writing, the purely motor subordinate center being located in the C_a cortex. In the analysis of the insula-central ganglion cases with agraphia, nine out of eleven showed an involvement of the external capsule. On the contrary, lesions limited to the lenticular nucleus or the insula showed no agraphia. One is justified, therefore, in the conclusion that the external capsule transmits association fibers for writing.

That this center for writing is a later developed center and developed through special training, there can be no reasonable doubt. On the ability to write depends culture and higher civilization. This depends on the normal functioning of a small area — the foot of the second frontal convolution.

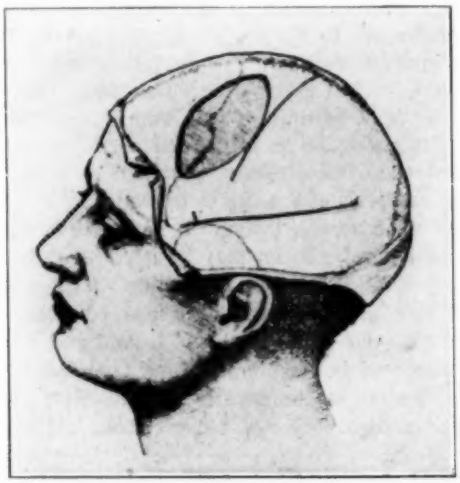

Fig. 6.—Starr's Case 1255, in which a tumor pressing on F_2 produced slowness of speech during two years before operation.

The Right Hemisphere.—It has been remarked by different observers — by Broca himself and by Charcot — that lesions of Broca's convolution are not always followed by aphemia, or that aphemia is but transitory. Moutier has assembled twenty-seven such cases. In fact, the localization of a motor speech center, or any other specialized speech center localization, could not be upheld were it not for the admission of a vicarious action of the right hemisphere.

The following reflections on this subject are pertinent:

1. Improvement or restitution of speech is not due to this function being taken over by an extended Broca's field. Many improved cases are those in which the lesion is limited to F_2.

2. In many cases, speech defects did not occur with F_2 lesions, or speech improved after such lesions.

3. In a few cases of acute trauma, the speech function remained undisturbed because of a rapid vicarious action of the right hemisphere.

4. In bilateral lesions of Broca's area, aphemia always exists.

5. Extensive lesions of the left hemisphere without aphemia and without aphasia of any sort when the entire speech field is involved, proves conclusively the vicarious action of the right hemisphere.

The child speaks as it is ambidextrous. Later there is a differentiation which determines whether the right or left hemisphere predominates. Stier's observation on a large number of German soldiers brought out the interesting fact that speech defects were four times more frequent in the left-handed. The right hemisphere seems to show a certain degree of inferiority as compared with the left.

In the sixth part, it was established that word deafness and word blindness might be compensated to a certain degree by the vicarious action of the right hemisphere, such as by hearing and reading without understanding (sinnlos). There is a great variability in this compensation, and in many cases, the right hemisphere cannot offer any restitution. The same general principles hold for motor speech. By destruction of the left Broca's convolution, many patients remain permanently aphemic owing to the inability of the right hemisphere to offer substitution. This is particularly the case, according to Mingazzini, when the lesion involves the white matter beneath the cortex of F_1 and attains the fibers of the corpus callosum. This authority holds that the right hemisphere is normally concerned with speech, but the words from this hemisphere are carried across the callosal fibers to the left hemisphere, which aids to form word images. This explanation, however, is not valid in those cases in which there is retention of speech with destruction of Broca's field and Mingazzini's area.

Transcortical Motor Aphasia.—The so-called transcortical motor aphasia, the existence of which is denied by Déjerine and by Moutier, and accepted by von Monakow, has many features in common with transcortical sensory aphasia. Henschen feels that there is a certain justification for the clinical picture, but no definite separate anatomic basis, and therefore no reason for defining it as a separate form of aphasia. The principal lesion is in F_3, or in F_2 in combination with midbrain lesions. It is, however, reasonable to suppose that repetition of words depends on integrity of the fasciculus arcuatus, also of the external capsule.

Apraxia.—Liepmann's epoch-making investigations of apraxia have thrown much light on variously described phenomena, described by Meynert as motor asymbolism. Liepmann in a late publication has asserted that motor aphasia is a particular form of apraxia of the glossolabio-pharyngeal musculature. This statement is further elucidated in a personal communication to Henschen: "Not all patients with motor aphasia suffer from apraxia, that is, apraxia of the extremities. An apraxia of the facial musculature, inability to whistle, to wrinkle the forehead, grimacing, etc., is certainly very frequent with motor aphasia. Apraxia of the hands, on the contrary, is relatively less frequent. There are certainly cases of motor aphasia, particularly those of pure motor aphasia, without apraxia, and conversely I have seen cases of apraxia without motor aphasia, or indeed aphasia of any sort."

The question arises whether the apraxia occurring in aphasia forms belongs to the aphemic or the dysarthritic cases. A statistical inquiry is rendered difficult because the term "apraxia" seldom has been used in case reports, and when used the special form of apraxia is not indicated. Mimicry is, however, often mentioned, and this term is used as an indicator of the presence or absence of a probable apraxia in aphemia, mutism, dysarthria and bulbar

paralysis, respectively. As pseudobulbar palsy and apraxia are often difficult to differentiate, as Liepmann himself has pointed out, Henschen offers his deductions with a certain reserve.

Unexpectedly, patients without apraxia showed a higher percentage of paralysis of the speech musculature than patients with apraxia. Some explanation of this paradox is in order, and it is suggested that mimicry in these aphasias is in the nature of an unconscious reflex act and does not correspond with the mechanism of more conscious speech. It would appear, therefore, that paralysis and amimia are different in origin.

Further, 428 cases of motor speech defect showed sixty-four of mimia and twenty-four of amimia. It seems justifiable to say, therefore, that apraxia and aphemia or dysarthria are different disorders and cannot be brought into any causal relationship.

Theories, Doctrines and Hypotheses.—Two main lines of endeavor have characterized aphasia research: the psychologic and the clinico-anatomic. Dr. Ernst Storch is one of the extremists of the constructive psychologic school. He has theoretically constructed a stereopsychic speech field in which there is a glossopsychic field, the disturbance of which occasions glossopsychic aphasia. Henschen criticizes this whole theory as "glosso-psychic phantasy."

S. Freud (1891) has advanced the hypothesis of a common speech field, of which the whole present work is an energetic protest.

Kurt Goldstein's conceptions are similar to those of Storch and Freud. Storch's stereopsychic field becomes to Goldstein a "concept field." His principal contentions are: the denial of any especial localization for the speech elements; the acceptation of a central associative speech field; and the placing of all aphasia forms in the cerebral cortex.

To Wernicke (1874) belongs the credit of localizing word deafness, although the discovery of this special form belongs to Bastian and to Kussmaul. Wernicke believed in a localization of only the elementary psychic functions such as sense perception; thought and consciousness was a function of the fiber tracts (Fasermassen). Memory pictures were classed as sense impressions. The three main forms of aphasia were: motor, sensory and conduction aphasia. Speech develops only in those who hear; thus mutism occurs in the deaf. Reading and writing were transcortical and subordinate to motor speech. Cortical motor aphasia from a direct lesion was followed by persisting alexia and agraphia. Likewise, these speech forms are lost when word comprehension or inner speech has suffered. A separate reading and a separate writing center were not recognized.

Although Wernicke was aware that certain contradictory cases to his constructive theories existed, these theories remained the same over a period of thirty years. In the foregoing pages, Henschen has repeatedly and completely refuted the ideas of Wernicke, which he believes have dominated aphasia research for many years.

Lichtheim followed Wernicke with his schematization, which included a new link—that of the concept center, which was connected to the motor and acoustic centers. As this scheme apparently explained the actual existing syndrome of the transcortical aphasia form, it became a great stimulus to the investigation of new forms of aphasia. Whereas it had a high didactic interest, it did not suffice for a clear explanation of other forms such as paraphasia and alexia.

Niessl von Mayendorf refutes the notion of separate perception centers and concept centers, which is without adequate anatomic or clinical basis.

Pierre Marie, in 1906, on the basis of the observation of 100 cases of aphasia, among which were fifty necropsies, found that his results did not confirm the classical doctrines of Broca. Three main assertions formed the basis of his thesis: Broca's convolution plays no especial rôle in the function of language; every aphasic presents more or less disturbance of comprehension; and there is an evident reduction of the intellectual capacity.

Every case of true aphasia is due to a lesion of Wernicke's field. This is often associated with a lesion of the lenticular region or "quadrilateral field," which causes in addition to aphasia, anarthria. Wernicke's aphasia plus anarthria equals aphasia of Broca. Inclusive in the aphasia of Broca neither word deafness, word blindness nor agraphia exists as a specific form. Marie's teachings are energetically opposed by Henschen, who denies that general intelligence defect is a consequence of aphasia, and who sees but a confusion of terms in the new nomenclature. He finds the term "quadrilateral field" poorly defined anatomically, and unscientific, and characterizes the whole as poor construction.

M. and Mme. Déjerine have contributed greatly to the problem of aphasia as well as to neurology in general. These authors are strong proponents of definite speech localizations, but Henschen differs from their views in three not unimportant points:

1. Pure motor aphasia, although clinically distinct according to the Déjerines, is not capable of an exact anatomic 'localization. To this view, Henschen cannot agree.

2. A lesion of the lenticular nucleus cannot produce aphasia. Henschen calls attention to his analysis that a lesion of the anterior putamen causes aphemia as a rule.

3. There is only one sensory form of aphasia comprising both speech comprehension and disturbance of reading. However, Déjerine recognizes two separate forms of word blindness; the ordinary form associated with agraphia, and a pure form without agraphia due to a subcortical lesion, affecting fibers from the right hemisphere. Henschen has established, on the basis of authentic case records, an independent form of word blindness due to a sharply circumscribed lesion of the angular gyrus. This fact is of fundamental importance. We learn to read by aid of audition, but the center for reading develops into a center quite independent from Wernicke's center.

Carl von Monakow makes a distinction between the localization of the aphasic symptoms and the location of speech itself; the latter has its seat in the whole cortex. Motor speech is placed in the extended Broca's field, namely, in addition to the foot of F_3, in the operculum, pars triangularis and the anterior part of the insula. In many respects, his views are similar to those of Marie.

Monakow uses his diaschisis theory in the evaluation of clinical aphasia symptoms which may tend to modify or disappear. It may therefore play the part of a rescuing angel rather than explain the exact mechanism.

In Norwich, in 1868, where Broca presented his views on aphasia, H. Jackson also presented his own theory. This was psychologic in trend. He said that there are two different forms of aphasia—the intellectual and the emotional. Head has followed the general point of view of Jackson. "He laid down in the discussion the fundamental basis of the whole of his future treatment of the subject. It is the power to form propositions that is affected and not the memory of words or faculty of language." In the historical presenta-

tion of his subject, Head asserts that at the end of the eighteenth century "No one presumed to imagine that the activities of the mind bore any direct relation to the life of the brain." This statement surprises Henschen, who points to the contribution of Galenus (200 A. D.) and Swedenborg (1740). The former, in discussing the functions of the brain, considered that the convolutions were not the seat of the higher intellectual faculties, because the ass presents a brain with convolutions.

The following quotations from Head[2] outline his views: "The faculty of speech is not localized in any area of the cortex; no lesion, however local, can affect speech and speech only. The cerebral injury disturbs certain physiological processes which subserve the complex acts which we speak of as speech. . . . There is no physiologic function or faculty corresponding to speech. We must therefore get rid of all *a priori* conceptions which underlie such terms as 'motor' and 'sensory' aphasia, 'alexia,' 'agraphia,' or 'amnesia verbalis.' . . . Still less can these hypothetical conditions be associated with limited destruction of any part of the brain."

Henschen's comment is as follows: With *a priori* conceptions he (Head) has made no analysis of aphasia material or of forms of aphasia, and his theory lacks any scientific confirmation and is therefore outside any scientific discussion. The influence of Head's doctrines is one of complete confusion.

Jendrassik and Bianchi have approached the problem of aphasia from the standpoint of localization—the former, on the basis of clinical and anatomic studies, is an extreme localizer. Bianchi has added the results of animal experimentation, and although differing in some points from Henschen, their views are in general the same.

Concluding Remarks.—The concluding chapter is "Schlussworte." Speech centers with sharp localization are the workshops of ideation (Begriffe). Ideation depends on concepts (Vorstellungen) of different associated auditory, optic, tactile, etc., nature. With the disturbance of the speech centers, not all concepts disappear—object concepts are generally conserved. There is much to support Bianchi's views that abstract ideation, which the word symbolizes and leads to expression, is however, affected. The number of stages to completion of word ideation from the auditory or optic word is not known. There are probably individual variations, but in the last the frontal lobe, and especially the left, is indicated as the final stage. The under surfaces of the temporal and occipital lobes are terrae incognitae. It is only by means of clinical and anatomic studies and of such researches as those of the Vogts, and of Brodmann, et al, that we may make progress in brain research. Through education and practice new centers and associated tracts out of previously unused cell material provide a new speech form, or new ideas (Ideenkrise). Brain centers are banded together, so that when one is affected the whole may be affected; thus in forms of aphasia, word blindness may occasion word agraphia, etc.

Education produces a high grade of picture imagery from the undifferentiated brain cell supply, and the centers thus formed as well as the association paths become habitual and inherited. Automatism plays an important part in the psychic life of the individual. The conscious life and new thought mechanism are thus relieved of a considerable burden. The right hemisphere is chiefly considered with this automatism, which is a psychic reflex.

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2. Head: Brain **43**:390-450.

COMMENT

This important work, accomplished after academic retirement, will stand as a monument to the learning and industry of its author.

As a statistical and analytic study of aphasia, the work stands unparalleled in the literature, and as a fund of information from almost every angle it is invaluable to the research worker.

It would seem rather presumptuous for any one but a leader and original worker in this field to offer criticism of a study that represents the mature opinion of an acknowledged master in brain research. We are a long way from finality in our knowledge of anatomy, physiology, or pathologic physiology of the brain, and it would seem that the safest plan at present is to work from established experience in brain pathology rather than to work from preconceived theories. This is the great contention of Henschen. He is a strict localizer as regards elementary brain perceptions and concepts. From the purely clinico-anatomic standpoint, Henschen appears to have established his point.

It appears to the reviewer that the whole situation hangs on the determination as to whether psychic processes are analogous to definite somatic (motor and sensory) functions with definite localizations, or whether they are something apart from them. The definite structure of the brain and all that is known of the function of organs, whether it be the heart or a lymphatic gland, leads us to the assumption that its function is definite, systematic, orderly and in accord with its structure. The other view would be either that the mind is apart from matter, which no neurologist believes today, or that dependent on matter (brain), the nerve impulses are in a state of flux; they may take this path or that according to the individual case, or in the same case vary according to incidental factors such as defect, inhibitions, lesions, intoxications, etc. Such ideas would commit us to a policy of indecision and hopelessness for future brain research. The fact that many precise brain mechanisms have escaped us up to the present time is not evidence that these mechanisms do not exist.

Some investigators who have been prominent in brain physiology have lately advanced ideas concerning the function of speech, which are chaotic in all that leads to clear thinking, rationalism or research. So much more honor to Henschen, therefore, to have stepped in at this opportune moment and to do what has evidently not occurred to the theorist, i. e., to examine the wealth of unexplored material on aphasia!

It would seem to the reviewer that Henschen is at times unnecessarily sharp in his criticism of those whose views are in contrast to his own. Such is the criticism of Marie. It does not seem just to call a neuropathologist who has contributed so much to clinical and anatomic knowledge of aphasia as being entirely "konstructiv."

Henschen's analysis of his statistics does not clear up the whole problem of aphasia. He has simply stated that certain lesions of the brain are as a rule followed by certain clinical findings. In this something fundamental has surely been achieved.

Abstracts from Current Literature

MULTIPLE SCLEROSIS. VERAGUTH, Rev. neurol. **31**:631-683 (June) 1924.

There are two main addresses on this topic in the pages enumerated. They are but a part of the Transactions of the fifth annual international neurological meeting held in Paris, May 30-31, 1924, under the auspices of the Society of Neurology of Paris. Almost the entire June number of the *Revue neurologique* is given up to the papers and discussion presented at this meeting. The first principal address is by Veraguth of Zurich on the general subject of multiple sclerosis. The second principal paper is by Georges Guillain, in which he presents a clinical, anatomopathologic and pathologenic study of the subject.

Veraguth begins his paper with a general statement as to the difficulties of diagnosis and consequently of prognosis. He comments on the inefficacy of therapeutic measures. He refers briefly to the history of research and study in this field, particularly that of Cruveilhier, Charcot, and of Pierre Marie, who was the first to write on the infectious character of the disease.

He states that Borst, in 1904, cited 252 German articles, eighty-four French and thirty-seven English devoted to multiple sclerosis. Since that time, numerous other articles have been written, probably already more than a thousand in number.

The writer first takes up the topic of diagnosis. Preliminary to this he refers to the discordance as to its frequency in different parts of the world. Switzerland alone is reported as having 1,500 persons with multiple sclerosis, or one in every 3,000 inhabitants, or a number greater than that of patients with syphilis of the central nervous system.

One difficulty in arriving at proper figures of incidence is that of the chance of error in mistaken diagnosis. This is due to the proteiform symptomatology. It is easy enough to recognize the Charcot triad, but cases showing this triad are rare. Veraguth says that among 150 cases that he has observed, the triad appeared in only 10 per cent.

The cerebrospinal type of multiple sclerosis presents the greatest difficulties in differential diagnosis, and is the object of numerous discussions. It is sometimes known as the acute form and is hard to distinguish from a disseminated encephalomyelitis. The early history of these two forms is much alike. It is not possible to be sure, in a case of multiple sclerosis, until successive remissions and recurrences are observed.

Marburg insists on the absence of fever in multiple sclerosis. Other authors contest this assertion. Veraguth believes he has seen cases with slight elevations of temperature explainable on no other basis than the disease itself. It is impossible to lay down any exact diagnostic criteria for a single clinical examination. More than a score of symptoms may appear, all the way from diplopia, paresthesias, lost abdominal reflexes, etc., to the Babinski sign.

In this differential diagnosis between multiple sclerosis and so-called encephalomyelitis, there may appear for consideration such clinical points as progressive amaurosis, astereognosis, dysmetria and sphincter disturbances, all accompanied by a negative Wassermann reaction. Where such a difficulty in diagnosis exists, it is necessary to follow the cases for a long time. Another more rare disorder which must be differentiated is that of poisoning from

manganese. The history will clear up the diagnosis. Another frequently difficult problem is to differentiate hysteria from multiple sclerosis. The most important evidence against hysteria is the Babinski sign.

The three following facts should always be kept in mind: first, the great toe sign may disappear temporarily in multiple sclerosis; second, there is nothing easier for an hysterical person to do than to reproduce the phenomenon of Babinski when he has been examined frequently in the company of persons with organic cases; third, hysteria may be associated with different organic disorders, and multiple sclerosis is no exception to this association.

So-called essential tremors are always a familial affection.

As opposed to the cerebrospinal form, there exists a so-called cerebral form of multiple sclerosis. In the latter, the predominance of isolated symptoms may result in diagnostic errors — such symptoms as temporal pallor of the nerve head, persistent scotomas (relative or absolute), passing diplopia, total unilateral, internal ophthalmoplegia and occasionally stiff pupils.

Some patients with multiple sclerosis present, at times, epileptic crises. They may be regarded as having cases of essential epilepsy until some time in the course of their examination they show the absence of abdominal reflexes and the presence of the Babinski sign, which persists long after the epileptic attack has subsided.

Berge has noted some cases of multiple sclerosis with festinating gait. This calls for differentiation from cases of Parkinson's disease.

Then there are instances of multiple sclerosis with hemiplegia, passing aphasia, etc. The author has also had occasion, because of the question of military insurance or pensions, to differentiate multiple sclerosis from post myelo-encephalitic sequelae. An exact history may determine the point. The patient may have shown an early somnolence of long duration. Such a history is against multiple sclerosis.

Two diseases of early childhood may be confused with multiple sclerosis (as we now know that multiple sclerosis is not confined to the second and fourth decades). One of these diseases of childhood is so-called tuberous sclerosis, a congenital disorder combined with idiocy and accompaniied by kidney and heart disturbances. The other is the so-called "malady of Pelizaus-Merzbacher," which sometimes shows nystagmus, intentional tremor and loss of abdominal reflexes. The latter disease is, however, always familial, occurring during the first six months in life and pursuing a rapid progressive course, with trophic involvement of the bony structures.

Confusion with Wilson's disease is not likely except in the first stages of the latter affection, in which nystagmus and atrophy of the optic nerve are not present.

Cerebral tumor is frequently mistaken for multiple sclerosis. Ventriculography may clear up the diagnosis. If it is a doubtful case masked by cerebellar ataxia, asynergy, adiadokokinesis, and Bárány's signs, it will be necessary to differentiate between multiple sclerosis and a paracerebellar or endocerebellar tumor. Further difficulties are encountered in patients with rigid pupils and disturbances from the seventh to twelfth cranial nerves.

Sometimes multiple sclerosis has to be differentiated from cerebellar atrophies. The latter can be distinguished by the slow evolution of the disease without remission and a predominance of disturbances of gait and of speech, the absence of spinal symptoms, pontobulbar symptoms and disturbances of sensibility.

Certain spinal types of multiple sclerosis demand consideration, the dorsal and lumbar varieties being more frequent than the cervical and sacral. According to Oppenheim, the cervical form begins acutely and is distinguished by the predominance of posterior column symptoms. The sacral type of multiple sclerosis may be confused with tumors of the cauda equina. Sometimes the sacral form in the male is accompanied by disturbance of sexual function. In such cases, it is necessary to rule out purely psychogenic factors.

The differential diagnosis between multiple sclerosis and certain forms of myelitis in which there is an insidious development without hyperthermia presents great difficulties.

The author then gives a brief resumé, in which he states that if the classic form of Charcot's disease be left out of consideration, multiple sclerosis in its various forms may simulate diseases most diverse and thus lead to frequent diagnostic criteria, namely: multiplicity of symptomatologic foci, an evolution marked by sharp remissions, absence of the abdominal reflexes, dysmetria and atrophy of the optic nerve. While these last named symptoms are found in the majority of forms of multiple sclerosis, they are associated in a variable fashion and may be entirely lacking. There remain, therefore, a number of types which force us to admit an absence of any specific sign that characterizes multiple sclerosis in its early stages.

The author gives an afterword to the studies of Guillain on the changes of spinal fluid in multiple sclerosis.

He also speaks of the works of one of his own pupils, Oberholzer, in the spinal fluid by the spectrographic method, first made notable by Victor Henri of Zurich. The latter found traces of fine inorganic substances in solution corresponding to the ultraviolet ray in concentrations of such a strength as could not be determined by methods of chemical analysis.

This particular approach to the study of multiple sclerosis is relatively in its early stages, but promises much and may lead ultimately to positive results. From the diagnostic consideration of multiple sclerosis, the author then passes to its pathogenesis. Instead of taking up the question of the exogenous or endogenous origin of the disease, he goes back to a more fundamental formula set forth by Strümpell, namely:

$$\text{Any Disease } \frac{\text{Real cause and the occasional cause}}{\text{Congenital resistance and acquired resistance}}$$

Following up such a formula, studies in multiple sclerosis have resolved themselves into anatomopathologic studies, clinical statistics, and works on experimental parasitology. As for the anatomopathologic approach, three opposing opinions are found in the literature:

First, the doctrine of endogenous primary gliosis has been defended by Ziegler, Strümpell and his school. This doctrine lays emphasis on an abnormal tendency to proliferation of the neuroglia tissue. The idea of acquired resistance, however, can find no place in such a theory. Those who accept the theory of endogenous gliosis believe that there are two varieties — essential primary and secondary sclerosis, the result of an inflammatory process. The favorite argument invoked in favor of a primitive gliosis is that the proliferation of the neuroglia is formed in all of the foci of multiple sclerosis, just the same as in those cases when the changes of the parenchyma or of the vascular tissue is present.

There are many other authors, however, who with Charcot see in the gliosis a primary process but inflammatory in character, analogous to interstitial inflammations such as may be found in the liver or the kidneys. Their principal

argument lies in the great frequency of the proliferation of the glia and in the presence of normal parenchyma between the fibrils of the neuroglia. They consider the vascular lesions as rare and unimportant. Those students who defend this point of view — the school of Vienna in particular — see in the demyelinization the primary fact.

The author considers the inflammatory theory the most plausible. It concedes that the infectious agent can attack separately the parenchymal neuroglia or the vessels, either separately or all together.

If these inflammatory hypotheses be compared with the formula noted above, it will be seen that they all present common characteristics; that is, a noxious cause sufficient to invade the neuraxis. The occasional causes favor its penetration into the organism. The congenital resistance will be divided up diversely between the mesodermic tissues and the two ectodermic varieties. This doctrine agrees implicitly with the idea of an acquired resistance appearing in the course of the disease.

An examination of statistical studies in a clinical field shows high frequency of multiple sclerosis which speaks also in favor of its infectious nature.

Nerve degeneration so widespread is hardly curable.

The author digresses for a moment to certain familial diseases, such as Friedreich's ataxia, Huntington's disease, Wilson's disease and familial atrophy of the optic nerve, remarking that multiple sclerosis in his experience can rarely be regarded as an hereditary disease.

If dependence can be placed on statistics, certain occupations seem to show a higher frequency, and the provisional results of the Swiss studies indicate a certain predominance in agricultural trades.

Strümpell's arguments against infectious origin of multiple sclerosis are quite strong. He contends that there is no proof of the transmission of the disease from one person to another. This is true, however, of other diseases of a definite infectious nature, such as epidemic encephalitis. The course of multiple sclerosis is other than that of ordinary infectious diseases. The same thing may be said, however, of a number of syphilitic affections of the neuraxis. We do not as yet know the focus of entrance of the infectious agent. Statistics do not help us on this point. In certain rare cases, multiple sclerosis is found combined with chronic tonsillitis but this proves nothing when we consider the thousands of cases of tonsillitis in which there is no evidence of multiple sclerosis. Steiner holds that the virus enters through the skin. His statistics, however, are not convincing. They seem to establish some degree of relationship, however, between the disease and its occurrence in a large number of those whose work is associated with trees and wood, as well as farmers. The idea of an intermediary host is somewhat seductive, but this is still not established.

As a consequence of the ideas of Pierre Marie on the infectious nature of multiple sclerosis, there arises the question whether this disease may not be the resultant of various infectious disorders or whether, on the contrary, it may not be due to a specific germ. Of late years, it has been thought that the disorder is due to a certain spirillum. Veraguth does not insist on this point, as he states his own attempts along this line have led to negative results. He contents himself with recalling that of all of the positive results obtained up to the present time, there are few which can stand against the criticism of bacteriologists as competent as Noguchi and Dorr.

The problem of the physiopathology of multiple sclerosis is dominated by the incongruity long noted between the anatomopathologic facts and the

clinical symptoms. Necropsy in a patient with many symptoms of this disease may reveal few pathologic foci, while some cases with few lesions may show a multiplicity of symptoms. Two questions therefore naturally present themselves, first: "Can we explain by their localization, isolated symptoms?" The answer is chiefly in the negative in spite of the fact that other cerebral disorders with scattered foci sometimes show symptoms identical with those of multiple sclerosis. The author mentions a case worked up by Chiray, Foix and Nicolesco in which there was a tremor of one half of the body of the type of multiple sclerosis due to a lesion of the rubrothalamic tract. The second question raised is: "How can portions of the brain still function when they are sclerosed?" The old answer that the axis-cylinder and the ganglion cells not being entirely destroyed could thus still perform their function, must be taken with a grain of salt. The more recent view is that there is a destruction of some of the axis-cylinders and their ganglion cells, but that others near them remain intact. The latter are sufficient to preserve function.

To explain the character of grave passing symptoms, the intermittent evolution and their remission is not easy on a purely topographical basis. It is wiser to suppose that there are different degrees of difficulties as a result of lesions in both those areas severely involved and in the intact tissues. The inherent difficulties in such explanations are removed if one accepts the theory of Monakow relative to "diaschisis" in multiple sclerosis. The great variability of clinical manifestations and of their uncertain evolution clears itself up in the light of this theory.

Circulatory disturbances in the nervous structure affects both the qualitative and quantitative nutrition of the neurons. The axis-cylinders may lose their sheaths, and the invasion may go as far as to the tissue of the myelaxis. Proliferation of glia may be so severe as to disturb mechanically the function of the nerve cells. Archucarvo has gone so far as to attribute to the glia, among other things, the function of an organ analogous to the internal glands. Referring again to the widely separated foci of lesions as well as to the variability in the degree of the intensity of the manifest symptoms, the author again reverts to the theory of diaschisis as accounting for the same. According to this theory, the consequences on the functions of the regions invaded are not only manifest, but also they exercise this effect on anatomically intact foci at quite a distance. This accounts for passing symptoms. The degree of the manifestation of disturbance in the intact foci depends in turn on the momentary state of their excitability.

Relative to therapy, the author's attitude is one of rather marked scepticism. He thinks the results reported in most cases are on the basis of either post hoc or propter hoc. In five of his own cases, he gave injections of calomel. Three of these patients gave him the credit of the cure. The other two as well as the authors, could see no apparent change. The injection of thiosinamin, or preparations of it is popular in certain quarters because it is accompanied with less distress. The author believes that enthusiasm of others in the use of arsphenamin and its derivatives is existent most largely among those who have treated few patients. He thinks that the studies of Mlle. Stern of Geneva have demonstrated conclusively that arsphenamin does not penetrate the hemato-encephalitic barrier. He thinks it less likely that arsphenamin penetrates the parenchyma where the infectious organism is formed.

The author devotes a paragraph to the matter of aids to therapy. One he attacks from a negative point of view. He believes there is danger in increas-

ing faith of women with multiple sclerosis in the efficacy of pregnancy and childbirth. He leans to the belief that most women suffering from multiple sclerosis should be sterilized.

Lumbar puncture, while it may aid in diagnosis, is accompanied with some danger. A few patients have developed a meningismus following puncture. In severe spastic cases, rhizotomy has proved of value, but in others, the patient has been worse off than before.

Any and all of these measures mean little to what may be hoped for if an antitoxin is discovered. This the writer regards as paramount. To Pierre Marie the glory for such an achievement would be deserving.

JONES, Detroit.

THE PATHOGENESIS OF CEREBRAL DIPLEGIA. JAMES COLLIER, Brain **47**:1 (Feb.) 1924.

Little, in 1861, first described the condition which the author discusses under the title of cerebral diplegia. Andry, Delpech, Heine, and others, under the name "cerebral spastic paralysis," and Joerg, in 1828, described some of the clinical features of cerebral diplegia. Little "established the incontestable fact that this condition is often associated with abnormalities of birth. . . . He assumed the affection to be a widely spread fine lesion of the cerebral substance, which could affect one part of the brain more than another. He stated the important fact that the less severe cases may show a progressive recovery in the course of years, thereby proving that slighter degrees of the lesion are recoverable. And lastly he excluded gross injuries to the brain during birth and gross lesions from the etiology of the disease which bears his name, and pointed out its common association with microcephaly." Little pinned his faith on asphyxia as the cause of the lesion of the central nervous system both in prolonged labor and breech presentations, and in precipitate labor and premature infants. While he studied meningeal hemorrhages in the new-born, he never suggested that meningeal hemorrhage is a cause of infantile spastic paralysis.

The author uses the term "Little's disease" only in those cases of diplegia associated with difficult birth. By the French school, the term is used for all conditions of cerebral diplegia present from the time of birth. Erb and Charcot, in 1875, described the clinical picture of primary lateral sclerosis in the adult, and this was soon followed by observations on cases occurring in childhood described as examples of "spastic tabes in childhood." These were really examples of the paraplegic form of Little's disease. Certain authorities looked on the lesion as being of spinal origin — E. G. Seligmüler, Foerster, Ruprecht, Naef — but the work of Wolters and James Ross proved the fact that the pathologic condition was cerebral. The lesion of the brain in cerebral diplegia was looked on as an atrophy and sclerosis of the convolutions, producing a walnut kernel type of brain. The lesion affected the rolandic area especially, and in the paraplegic forms was chiefly noticeable in the paracentral regions. This was the "lobar atrophic sclerosis" of the French and was thought to be the result of some previous pathologic process, the nature of which was unknown. It was pointed out also that the paralysis might be severe and the atrophic sclerosis slight.

In 1885, Sarah MacNutt described her cases of "double infantile spastic hemiplegia" and, on the basis of three cases, concluded that meningeal hemor-

rhage is the universal cause of infantile spastic states dating from the time of birth and associated with difficult labor. "This opinion expressed by her has gained the widest acceptance, so that one reads on every hand that meningeal hemorrhage is a proved cause of diplegia. Yet her case of diplegia did not date from birth, for upon the anatomical findings the disease must have been installed early in fetal life, and her case of meningeal hemorrhage was not the result of difficult labor, for the birth is described as having been particularly easy. It was not a case of meningeal hemorrhage, but an intercerebral hemorrhage. . . This cerebral hemorrhage did not occur at the time of birth, but on the twelfth day after birth. . . Yet this is the evidence upon which the case for meningeal hemorrhage as a cause for diplegia rests. There is no other evidence. Freud, in his magnificent monograph of 1897, searched clinics, museums and the literature in vain for other evidence and found none. And that, too, has been my experience. The French pathologists have never accepted meningeal hemorrhage as cause for diplegia. . . The evidence demands the verdict that meningeal hemorrhage should be deleted as a causal factor of any infantile spastic state."

Strümpell, in 1885, brought forward undoubtable cases of postnatal diplegia and infantile hemiplegia due to polio-encephalitis. He argued this initial pathology for infantile spastic states in general, but there does not seem to be much evidence for fetal polio-encephalitis. Virchow, in 1865, however, described "encephalitis congenitalis," and von Linebeck considered atrophic lobar sclerosis to be the result of this condition. Recently, others have shown that encephalitis may occur in intra-uterine life, so that this cannot be refuted altogether. "All recent evidence has shown that polio-encephalitis is a common cause of post-natal infantile hemiplegia, and that acquired diplegia with severe initial symptoms may also be of this nature." Other forms of encephalitis can produce the clinical picture of diplegia, both in childhood and adult life; for example, encephalitis after specific fevers or epidemic encephalitis.

In 1894, Brissaud brought forward the theory that prematurity of birth was the essential causal factor in cerebral diplegia, and stated that birth before time arrested or retarded the essential functions of evolution of the fetus. His evidence is based largely on the authority of Flechsig, who asserts that the pyramidal system commences to grow out of the cortex in the fifth month and is only completely developed at the age of three and one-half years. He says "what takes three weeks to grow in intra-uterine life will take it three years or never in extra-uterine life in the prematurely born, and thence arises the spastic paralysis." The author points out that most premature children have completed the growth of their nervous systems and most of them do not develop Little's disease. Brissaud was supported in his argument by Marie and Van Gehuchten.

"In 1897 came the grand monograph from Freud, of Vienna." He had collected material for ten years and published his volume "Infantile Cerebral Paralysis," which is a collection and review of all preceding authors, together with reports of his own cases. This book "still remains the most complete and authoritative exposition of the subject." Freud argued that all the diplegias which dated from birth, and which had been attributed to anomalies of birth, really had their pathologic origin long before birth in intra-uterine life. He disproved the "meningeal hemorrhage" hypothesis. He piled up statistics to show that 40 per cent. of all diplegias dating from birth were without any possible factor connected with birth, neither premature, precipital,

prolonged or instrumental birth, nor asphyxia, or postnatal convulsions. He sums up his views as follows: "Premature, precipitate and difficult birth and asphyxia neonatorum are not causal factors in the production of diplegia; they are only associated symptoms of deeper lying influences which have dominated the development of the foetus or the organism of the mother." The only cerebral paralysis which results from difficult labor occurs when the brain is lacerated, and this takes the form of a monoplegia or hemiplegia, rarely a diplegia, and the anatomic findings are very different from those in the brains of infants with diplegia. Freud then goes on to prove the presence of a primary neuronic degeneration in postnatal cases of diplegia, the degeneration being incident during any period of fetal life or during childhood.

In 1909, Auglade and Jacquin reported a case of spastic diplegia with necropsy findings with a neuronic degeneration of the cerebellum in addition to a similar and more extensive condition in the cortex of the cerebrum. This was the first record of primary involvement of the cerebellum in diplegia.

In 1908, McCarrison described what he called nervous cretins who were myxedematous idiots with typical cerebral diplegia. One third of all his cases were of this type. The diplegia seemed to develop toward the end of the first year of life, and was attributed by McCarrison to a toxic process from a deficiency in the thyroid and parathyroid function in eliminating the toxins produced in normal metabolism. In 1913, Chagas reported a case of infection with *Trypanosoma cruzi,* with symptoms closely resembling cerebral diplegia. Others have reported cases of nervous cretinism similar to those of McCarrison. "McCarrison undoubtedly proved the clinical association of diplegia with cretinism in his endemic cases. He did not prove that the diplegia was the result of thyroid disease, for it is justly argumentable that both the cerebral and the thyroid lesions in his cases were two separate effects of one common infection."

The author then reviews the etiologic factors of diplegia. Hereditary and familial influences are exceptionally rare in the common type of the disease. Oppenheim reported a case of a mother who was the subject of a prenatal diplegia giving birth to a child with the same affliction. Familial incidence in prenatal and postnatal cases have been reported.

Maternal ill health during pregnancy is a matter of little importance in the etiology of cerebral diplegia. Gowers pointed out that many infants with diplegia are the first born of healthy young mothers. Syphilis may be a causative factor.

Abnormalities of birth are excluded by the author as causal factors, and are placed by him as common diplegia, indicative of something wrong with the fetus.

What deductions can be drawn from the pathologic material? In the first place, arrest of development due to an affection of the brain during fetal life and not to any events connected with birth is to found in all cases of diplegia in which symptoms are present from the time of birth. The first signs of this affection are expressed in neuroblastic and neuronic death or degeneration. "The one anatomical lesion which has been found in all cases of diplegia since it was first pointed out by Cotard in 1868, is disappearance or degeneration of the neurons of the brain. I hold that this is a primary degeneration of the neurons, and that it is the essential and primary lesion in all cases of diplegia. Atrophic sclerosis of the brain is the natural consequence of the neuronic decay."

ALPERS, Philadelphia.

CONGENITAL DIPLEGIAS AND THYROID DISTURBANCES IN ABNORMAL INFANTS OF GENEVA. F. NAVILLE, Schweiz. Arch. f. Neurol. u. Psychiat., Festschrift für Constantin von Monakow **13**:559-567, 1923.

A number of writers have stated recently that certain cases of spastic congenital or acquired diplegia may be due to dysfunction of the thyroid gland, and may be cured by the administration of thyroid preparations.

Three causes may act to produce congenital spastic diplegia. 1. Encephalic or medullary lesions dating from the fetal period, such as agenesis, malformations, vascular, infectious, traumatic lesions, etc. 2. Hemorrhagic lesions due to pathologic delivery, such as compression, asphyxia and obstetric traumatism. 3. Agenesis of the pyramidal tracts in cases of premature birth.

It was not until recently that it was thought that thyroid disturbances could be held responsible for true spastic diplegias. Support of this view is found in the improvement of those patients on the administration of thyroid preparations, although it must be borne in mind that Little's disease may regress spontaneously without treatment. Thyroid medication does not alter an established case of Little's disease, but it does benefit those cases diagnosed as Little's disease, in which the limbs are feeble, and in which tetany and spasms appear. The parathyroid body does not give these results.

In order to form a personal opinion regarding this matter, Naville reviewed the cases seen by him during twelve years of sanitary service for the schools of Geneva. There were 164 cases as follows: 1. In twenty cases of acquired diplegia, disturbances of the thyroid body were not in question. These cases resulted from syphilis, traumatism, acute infections, etc. 2. There were forty cases of simple myxedema, but without any diplegic motor disturbance. 3. There were twenty cases of birth palsy. Here it was difficult to decide that birth was the only active cause in the production of the diplegia, and that some previous malady or malformation might not have been responsible. The fact that birth is complicated by prolonged asphyxia is no proof that the malady is of recent acquisition. 4. There were sixty cases of congenital diplegia, neither obstetric nor dysthyroidgenic, but due to fetal disease or malformation. This group comprised most cases of congenital diplegia. The polymorphism of these cases was extreme and included quadriplegia, paraplegia, dysarthria, aphasia, disturbances of intelligence, convulsive seizures and psychomotor disturbances. Not infrequently these were associated fetal malpositions, osseous, genital and lens deformities, spina bifida, obesity, dystrophy, hemiatrophy, hydrocephalus, cranial rickets, oxycephaly, prolonged digestive disturbances, and disturbances in motor speech and intellectual development. Unfortunately, no laboratory tests could be relied on to establish the question of thyroid disturbances in doubtful cases. 5. Only twenty-four cases remained in which it seemed possible to impute the disorders of motility to a thyroid disturbance. Eight of these cases had to be put aside since thyroid disturbances alone would not explain them. In sixteen cases, it seemed certain that dysthyroidism bore a probable relationship to the encephalic disturbance. Twelve of these, however, did not have true diplegia, or at least not so at the age at which he observed them. They presented only some of the signs of thyroid disturbance, such as cretinoid facies, slight cutaneous myxedema, goiter or an absence of the thyroid, general dystrophy, psychomotor apathy, etc. Thyroid disturbances were certainly responsible for some of the symptoms noted, but it would be difficult to state which.

Of 1,700 infants between 5 and 8 years of age examined for admission to the classes of abnormal children in Geneva schools, there were twenty cases not related to thyroid disturbances, forty cases of myxedema without neurologic symptoms, eighty cases of congenital diplegia not based on thyroid disturbances but based on fetal cerebral lesions, eight cases of cerebral and mental disturbance of an indeterminate nature, but of which none could be attributed entirely to thyroid trouble, twelve cases of cerebral disturbance, probably dysthyroidgenic, but not true diplegia, and only four cases in which thyroid disturbances were associated with a true congenital diplegia.

In these cases, it seemed that the thyroid disturbance was present during the fetal period and not only after birth. This probably brought about a perturbation in development and an arrest of growth at an early state, and explains the difficulty of therapy and the importance of the prophylactic administration of iodin to the entire population in which endemic goiter exists.

WOLTMAN, Rochester, Minn.

HYSTERICAL PAIN. R. G. GORDON and H. H. CARLETON, Brain **46**:221 (July) 1923.

Babinski and Hurst maintain that the hysterical symptom is the product of suggestion. The psychanalytic school declares that the hysterical symptom is always a symbolic manifestation of an underlying psychic complex. The authors believe that the hysterical symptom is the product of suggestion and a well defined complex, and that these determine the localization and character of the hysterical symptom. They declare, moreover, that dissociation from cortical control is characteristic of all forms of hysterical symptoms, and this is particularly true of hysterical pain.

"Hysterical pain is a release phenomenon in which, owing to functional dissociation of cortical control, the more primitive sensory system convergent upon the optic thalamus is unmasked and holds sway."

The etiology and nature of hysterical pain are difficult to determine, because pain is subjective. If a patient complains of pain, can we say it is all imagination, and that he feels no pain? According to certain authors, we should never dismiss an alleged pain as imaginary or simulated until the possibility of an organic cause has been excluded. While pain has two sides, affective and cognitive, it is probably largely affective, and this affective side has much in common with the feeling of unpleasure. This unpleasure, as Freud points out, represents a state of extreme physical tension or instability, and this is characteristic of phenomena of nervous activity released from cortical control. Hysterical pain may be pain which is bound up with this primitive feeling of unpleasure (unlust).

"Hysterical pain may be referred to any part of the body, and may be described in as many different terms as there are qualifications to the word pain. These pains will, however, be affective in character, that is to say, their localization will be inexact and their intensity will be out of all proportion to the stimulus arousing them."

Headache.—There are no striking features pathognomonic of hysterical headache; but speaking generally, it is affective in character and vaguely localized; its severity depends largely on the attention it claims and bears no relation to the intensity of the stimulus, if indeed any stimulus can be discovered. In diagnosis, the process of elimination and considerations of probability must be employed. The psychogenic origin is to be found in

suggestion and the interaction of the latter with a complex, while the physiologic explanation lies in synaptic dissociation at or near cortical levels, which unmasks the primitive sensory mechanisms of the thalamus.

Painful Scars.—In cases of hysterical pain in relation to scars, there are two types of cases: (1) cases in which suggestion plays a large part and (2) cases of hysterical perpetuation of pain, originally of organic origin. Both types are presumably due to cortical dissociation and show the characteristics of enhanced thalamic activity. While patients with the first type of case are sometimes treated and cured by countersuggestion, there is another form of treatment which is quite successful in both types of cases. This form of treatment consists of hyperstimulation of the painful skin area involved. Before treatment, the fact that the brain is involuntarily blocking the stream of sensation from the affected area which he dreads is explained to the patient. Hyperstimulation is then applied to the area affected, consisting of rubbing, kneading, faradic current, smacking with the palm of the hand or with a strap, etc. The authors have treated thirty-eight patients with success by this method.

Visceral Pain.—This belongs to the protopathic thalamic system (Head), and in consequence its reference is vague and its intensity extreme. It is impossible to diagnose whether pain is hysterical or not from the type of pain in visceral pain, "since all such is thalamic in character, and the diagnosis can only be by inference, both negative and positive;" that is to say, by the absence of other symptoms and signs which should be present were the lesion organic, and by the recognition of some foregoing suggestion such as would be likely to determine the pain as a hysterical symptom.

The pain which results from hysterical contractures is not hysterical pain but organic, according to the authors.

ALPERS, Philadelphia.

UREMIA AND THE CHOROID PLEXUS. P. VON MONAKOW, Schweiz. Arch. f. Neurol. u. Psychiat., Festschrift für Constantin von Monakow **13**:515-525, 1923.

In a previous communication, the author expressed the opinion that the changes noted in the choroid plexus of patients who died of uremia are probably not without significance. It is usually stated that the cause of uremic coma is to be found in a toxic condition of the blood resulting from renal insufficiency. This view does not take account of any mechanism the organism may have of protecting itself against such substances. There is no doubt that these same blood changes may exist for a considerable length of time before coma sets in, and that uremic coma may appear precipitately without any manifest increase in the degree of renal insufficiency or change in the blood chemistry.

Uremic coma certainly is not alone dependent on the presence of toxic substances in the blood even though these are presumed to be necessary. The analysis of tissue fluids in cases of edema has revealed the presence of metabolic products which have been deposited in the tissues; such an edema may persist for a long time without the production of coma. Either the retention of these products, even in considerable amounts, has no influence on the brain, an assumption that is unlikely, or these substances are potentially toxic, but are held in abeyance by some mechanism which protects the brain from them. This mechanism includes a number of structures which reinforce each

other in the following order: choroid plexus, ependyma, glia membrane and cell membrane. Goldmann has shown that certain dyestuffs when injected intravenously will produce an intense staining reaction in the choroid plexus and in parts of the arachnoid, but they do not reach the ventricular fluid. Stern has shown that the plexus epithelium acts as a selective barrier in that some substances, such as bromides, salicylic acid, morphin and epinephrin pass through the choroid plexus and may be demonstrated in the brain, whereas others, such as iodin, arsphenamin, and trypan blue do not reach the ventricular fluid and cannot be demonstrated in the brain. Certain metabolic products, such as urea, pass through the plexus without difficulty, whereas others, like uric acid, creatinin and indican, do not or do so only slightly.

The function of the ventricular fluid has long been a subject of debate. By some it is said to serve as a buffer. In contradistinction to these mechanical views, C. von Monakow has advanced the theory that the ventricular fluid has as its chief function the nutrition of the brain, and that it is furnished by the choroid plexus as needed, passing through the ependyma into the brain, and through the brain by the way of His' spaces into the subarachnoid space, and thence into the lumbar sac. A direct communication through preformed openings is denied; the ventricular fluid is separated everywhere from the subarachnoid fluid by a membrane, the foramina serving only to allow choroidal vessels to pass through it. The question now arises as to whether the selective action of the choroid plexus is related to the appearance of uremic coma, and whether a failure in function of this structure may not be held responsible for the appearance of uremic symptoms, since the toxins circulating in the blood apparently have not altered in character or in amount. Tannenberg found changes in the choroid plexus in six of seven patients who died of uremic coma. He also found changes in the choroid plexus in cases of diabetic coma, eclampsia, hydrocephalus, status epilepticus, and tuberculosis. Von Monakow examined the choroid plexuses of rabbits from which both kidneys had been extirpated. The plexus epithelium was found to be swollen, vacuolated and hemorrhagic.

WOLTMAN, Rochester, Minn.

THE EARLY CLINICAL DIAGNOSIS OF METASTATIC CARCINOMA OF THE SPINAL COLUMN WITH A REPORT OF TWENTY-ONE CASES. D. C. WILSON, Clifton M. Bull. **10**:15 (March) 1924.

Symmers found 298 cases of malignancy among 5,155 necropsies at Bellevue Hospital, of which 74 per cent. showed metastases. Fraenkel found metastatic carcinoma in the spinal column in 20 per cent. of his cases. Pfahler found bone metastases in thirty-five of his cases, twenty-five of which involved the spinal column. He describes an osteoclastic or destructive type and an osteoplastic or slowly growing type where there is increase in the density of the bone. These types are accepted by all other writers, one type rarely being found without the other. The osteoplastic type usually arises from the prostate and the osteoclastic type from other sources. Blumer found bone metastasis in 2.9 per cent. of the cases of carcinoma of the uterus, in 2.6 per cent. in cases of carcinoma of the stomach, in 2.7 per cent. of esophageal cancers, in 25 per cent. of kidney cancers, in 36.9 per cent. of thyroid cancers, in 16.5 per cent. of breast cancers and in 70 per cent. of prostatic carcinoma. He classifies bone meteastases into five types, one of which is the spinal type. The latter is of two kinds, the nerve root type with excruciating pain, depending

on the location of the growth, and the paraplegic type which follows the preceding after months of suffering and is followed by paralysis of the body below the site of the lesion.

The author studied twenty-one cases of metastatic carcinoma to the spine; eleven occurred in males and ten in females. In the females, the breast was the location of eight of the primary growths, the mediastinum in another, and the location of the last was not determined. In the males, the prostate was the source of the primary tumor in eight, the liver in one, the stomach in another and the kidney in another. All of the patients complained of pain on admission except four. The pain was usually located in the back and radiated down the leg, simulating sciatica. Few patients had definite root pains. "The physical findings were practically of no value in diagnosis except in that they showed the location of the primary tumor, secondary anemia and spasm and tenderness of the muscles of the back." These findings were not constant but occurred in the majority of the cases. The reflexes were definitely abnormal in three cases, and in three others they deviated from the normal slightly. In only two cases were there sensory changes, these being anesthesia to light, pain and touch. Roentgen-ray examination showed ten of the spinal lesions to be osteoclastic in type. All of the diagnoses were confirmed by roentgen ray and five were proved by necropsy.

"The early diagnosis of this condition is very difficult. The outstanding clinical findings are first, pain in the back and legs which may in no way suggest cord pain; second, tenderness and spasm of the structures over the lumbar spine and sacrum; third, secondary anemia often of very mild degree; fourth, a moderate loss of weight. . . . The roentgen-ray is the chief source of information and should be used to study the spine of any patient in the fourth, fifth or sixth decade who complains of constant pain."

ALPERS, Philadelphia.

THE COELIAC REFLEX. ANDRÉ-THOMAS, Schweiz. Arch. f. Neurol. u. Psychiat., Festschr. f. Constantin v. Monakow **13**:617-621, 1923.

Reflexes of the vegetative nervous system cannot be obtained with the same regularity as those whose mechanism lies in the cerebrospinal axis, and their interpretation is extremely difficult. The pilomotor reflex gives us interesting information concerning the sympathetic nervous system, and aids in the diagnosis of a considerable number of diseases. The methods of eliciting this reflex will doubtless be perfected and its physiologic significance better understood.

The clinical value of vegetative reflexes is not as apparent as is that of the cerebrospinal reflexes. It is for this reason that Thomas again calls attention to a reflex described by him in collaboration with J. C. Roux. The following is essentially what is meant by the celiac reflex. When in the course of palpation of the abdominal wall the hand is pressed into the middle of the epigastric region, the radial pulse will be found to become less strong, and in some subjects it will disappear entirely. It is not necessary to conduct this experiment with great energy or to produce pain. In some cases, alternate pressure and relaxation will be accompanied by a corresponding fluctuation of the pulse. The phenomenon is most marked when the pressure is strong and painful. The duration of the pulse alteration is variable; it may persist as long

as pressure is continued, or it may disappear before the pressure is relieved. When there is difficulty in detecting changes in pulse with the finger, a pneumatic cuff may be applied to the arm using the minimal inflation necessary to produce obstruction to the passage of the pulse, or else a tracing may be made.

The reflex is found often, first, in anxious neuropathic patients and in patients with melancholia with a complaint of dyspesia; secondly, in patients whose epigastric sensitivity is already well marked; and thirdly, in diseases of the gastro-intestinal tract. It may at times be obtained in persons who fit in none of these categories.

It would seem that this reflex is dependent on an excitation of the celiac plexus, and that it is brought about by cardiac action. Guillaume interprets the phenomenon on a mechanical basis, assuming that compression of the abdominal aorta results in a disturbance of the circulation. The reflex was found present in twenty-four of twenty-nine cases of chronic appendicitis. In four of these cases, the reflex was absent, and once there was an augmentation of the radial pulse. The appendicular reflex, which is scarcely different from the celiac reflex, appeared after the injections of pilocarpin and disappeared after the injection of atropin. Claude and Tinel and Santenoise studied the reflex in conjunction with the oculocardiac reflex, and came to the conclusion that one is the inverse of the other. The cardiac reflex was absent in those subjects in whom the oculocardiac reflex is most accentuated. The diversity of response of the celiac reflex depends not only on the predominance of one or the other of the vagus or sympathetic systems, but also on general and temporary states of excitation of the subject. More prudence is required in the interpretation of visceral reflexes than in the interpretation of reflexes mediated through the cerebrospinal system.

WOLTMAN, Rochester, Minn.

ON THE RHINENCEPHALON OF DELPHINUS DELPHIS. WILLIAM H. F. ADDISON, J. Comp. Neurol. **25**:497 (Oct.) 1915.

The brain of the common dolphin is characterized by the absence of the olfactory tracts and bulbs; hence, the dolphin is completely anosmatic. Broca, in 1878, first divided *Mammalia* into osmatic and anosmatic groups according to the relative state of development of their olfactory apparatus. Turner, in 1890, further subdivided these into macrosmatic, microsmatic and anosmatic forms, apes and mammals being microsmatic and dolphins anosmatic. It is not known whether anosmatics have an olfacory apparatus in fetal life, but they are lacking in one in adult life. The brain of the dolphin was studied in this case to determine the extent of the regression.

It was found, on study of the brain of the dolphin, that in addition to the lack of olfactory bulbs and tracts, the olfactory cortex of the basal surface of the frontal lobe is also wanting. The result is the presence of a conspicuous oval on each side, called the *lobule desert* where the cortex is lacking and where the head of the corpus striatum comes to the surface. The parolfactory cortex is also much reduced, but at least some definite remains of it are seen. "Of the several connections of the olfactory and parolfactory cortical cells with the hippocampus, none were seen with certainty." Both the stria medullaris thalami (taenia thalami) and the taenia semicircularis are seen, as are their respective end stations; the ganglion habenulae are

very degenerate small structures; the fissura hippocampé is shallow but definite, and the subiculum is present. Connected with the hippocampus is the fimbria, seen as a slender band of fibers. True fornix fibers are seen and can be followed in sagittal series of sections. The corpora mamillaria are greatly reduced in size and the psalterium, formed of crossing fibers between the two hippocampi, is present distinctly. One septum pellucidum is very thin.

"Thus, the study of the brain of the dolphin shows that great atrophy of the hippocampal formation accompanies loss of the external olfactory structures, and that practically all the connecting tracts are likewise suppressed. The fact that both nucleus amygdalae and ganglion habenulae are present, though somewhat reduced, points to their having only a partial connection with the olfactory mechanism. There is a slight persistence of the parolfactory cortex."

ALPERS, Philadelphia.

VISUAL DISTURBANCES PRODUCED BY TRYPARSAMIDE. A. C. WOODS and J. C. MOORE, J. A. M. A. **82**:2105 (June 28) 1924.

The visual disturbances resulting from the administration of tryparsamide fall into two general classes, which the authors have designated as subjective and objective reactions. The subjective type is without definite ocular findings or visual field changes when examined at the height of the reaction or at any other time. The type classed as objective constantly shows changes in the visual fields and occasionally diminution of vision.

The gross incidence of untoward ocular symptoms in their 241 cases was 17.8 per cent. Twenty-four cases, or 10.2 per cent., fall in the subjective group; thirteen, or 5.5 per cent., in the group with objective ocular damage; and in five, or 2.1 per cent., the influence of tryparsamide is questionable.

The subjective reaction strongly suggests hyperesthesia of the retina as the underlying cause. From six to twenty-four hours after the injection of tryparsamide, a "dazzling" sensation appears, at times associated with a "tremor in the air." In this group, ophthalmoscopic examination gives negative results.

In the type of reaction termed objective, the same symptoms of dazzling vision are present. The symptoms appear, as in the subjective group, about fifteen hours after the treatment with tryparsamide, and are usually accompanied by a slight dimness or veiling of vision. In this more intense type of reaction, patients constantly stated that objects in a dim illumination appear gray. Vision frequently shows a slight but definite diminution. The visual fields show a noteworthy alteration, consisting of a concentric contraction of the form fields, affecting particularly the nasal, upper and lower fields, the temporal field being involved last or not at all.

In a few of the more severe cases, visual failure and field contraction slowly increased to a maximum within three weeks, in every instance except one, however stopping well short of blindness. The reaction progressed in this one case to practically complete blindness, and, within a period of two months, there appeared advanced primary optic atrophy.

Ninty-four per cent. of all reactions occurred early in the course of treatment, by the time of the tenth injection. Disease of the central nervous system, especially general paralytic and tabetic neurosyphilis, is to some extent a predisposing factor to visual disturbances from tryparsamide.

NIXON, San Francisco.

THE DEVELOPMENT OF THE PURKINJE CELLS AND OF THE CORTICAL LAYERS IN THE CEREBELLUM OF THE ALBINO RAT. WILLIAM H. F. ADDISON, J. Comp. Neurol. **21**:459 (Oct.) 1911.

The *outer* granule layer forms the outermost layer over the entire cerebellar cortex of the albino rat, from the second day before birth to the third week of prenatal life. From birth onward, it is composed of an outer layer of round cells and an inner layer of parallel fusiform cells. The layer increases in thickness up to eight to ten days after birth, and is then composed of from eight to ten rows of cells.

The Purkinje cells are easily found at birth along the inner boundary of the molecular layer. During the first week, there is great increase in size of both nucleus and cytoplasm in these cells. At from eight to ten days, there is definite change in form by the elongation of the cytoplasm of the ectal pole to form the dendrite. At the same time, all the dendrites become arranged in one plane, and this plane is parallel to sections directed across the folia. Nissl granules appear in the cytoplasm at from eight to ten days. The arrangement of the Purkinje cells changes with the increase in the surface area of the cortex. At birth, they are arranged in two or three irregular rows; at three days, in one or two irregular rows, and at five days, in one continuous row. As growth of the cortex continues, the space intervening between the Purkinje cells becomes greater.

The molecular layer is at first a narrow zone. Then, coincident with the growth of the Purkinje cells at from eight to ten days and the reduction of the outer granule layer, the molecular layer widens at a rapid rate until the middle of the fourth week.

The cells of the inner granule layer are derived from the outer granule layer and the mantle layer. The layer increases slowly during the first week of life. The grouping together of the cells of the granule layer with cell-free spaces between is already beginning at twelve days and is distinct at twenty days.

"The development of the motor activities of the young rat is closely correlated with that of the cerebellum and the animal is in full possession of its motor powers when the cerebellum has attained its mature arrangement."

ALPERS, Philadelphia.

POSTENCEPHALITIC BEHAVIOR DISTURBANCES WITHOUT PHYSICAL SIGNS. BERT I. BEVERLY and MANDEL SHERMAN, Am. J. Dis. Child. **27**:565 (June) 1924.

The authors present two cases in which the patients developed behavior disturbances of a postencephalitic type prior to the development of physical signs. In both instances, parkinsonian syndromes appeared; in one, six months after examination, in the other, two years after examination.

CASE 1.—The patient was a boy, aged 11 years. The difficulties in behavior are noted as "disobedience, lying, fighting, stealing, and cruelty to children and animals." The change in conduct began rather abruptly about three months after an attack of what appeared to be encephalitis. At the time of the first examination, the neurologic examination was negative; six months later, parkinsonian signs developed.

CASE 2.—The patient was a boy, aged 15 years. The medical history, except for frequent colds, was negative. At the time of the first examination he had become "incorrigible and a great nuisance to teachers." The physical examination was negative except for slightly irregular, unequal and sluggish pupils. Two years later, a typical parkinsonian syndrome developed.

VONDERAHE, Cincinnati.

HEMIANOPIA AS THE SOLE CLINICAL FEATURE IN UNTREATED SECONDARY SYPHILIS. A. W. S. SICHEL and A. R. FRASER, Am. J. Syph. **7**:665 (Oct.) 1923.

Involvement of the nervous system in syphilis is an early occurrence in the condition, and "it is highly probable that generalization of syphilitic infection has occurred before the chancre appears." Hence, central nervous system symptoms early in the disease in certain cases should not cause surprise.

Hemianopia is not a rare manifestation of syphilitic meningeal affection. It may be found in cases in which the optic radiations are affected, the lesion occurring anywhere between the chiasm to the occipital lobes. This symptom, however, is usually a late one, due to endarteritis, perivascular infiltration, and gummatous changes. Oscillating hemianopia (hemianopia fugax) has been described as characteristic of basilar syphilis. Other visual changes occur in syphilis. "Such changes as choked disc, optic neuritis, irregularities in the field of vision, scotomata, optic atrophy, etc., are well recognized as being late luetic stigmata."

The authors report a case of hemianopia in an untreated secondary syphilis, appearing about five months after the primary infection. The visual trouble was described as a right homonymous hemianopia. There was complete absence of systemic syphilitic manifestations.

ALPERS, Philadelphia.

CONCERNING THE CORTICOTHALAMIC RELATIONS IN THE MOTOR ZONE OF A RABBIT. JOSÉ M. DEVILLAVERDE, Schweiz. Arch. f. Neurol. u. Psychiat., Festschrift für Constantin von Monakow **13**:665-674, 1923.

The individualization of different fields of the cortex on a cytoarchitectonic basis suggested an attempt to determine the relationship that might exist between each of these zones and the various nuclei of the thalamus. The Marchi method made this possible. In line with this, the author attempted to establish the relationship between Broadmann's field No. 4 in a rabbit and the thalamus. He cauterized this area, which appears at the upper and anterior margin of the hemisphere, permitted time for degeneration and prepared serial sections. This field in the rabbit is analogous to the field containing the giant pyramidal cells in man. DeVillaverde found that it bore a definite relationship to the anterior nucleus of the optic thalamus, in addition to the projection system going to the internal capsule, and certain other relationships which did not concern the author in this connection. He was unable to establish a relationship to other portions of the thalamus. The fibers going to the sensory ganglion, described by Cajal, who used the Golgi method, must take their origin in other parts of the cerebral pallium.

WOLTMAN, Rochester, Minn.

THE SKIN LESIONS IN MENINGOCOCCUS SEPTICEMIA. C. L. BROWN, Am. J. Dis. Child. **27**:598 (June) 1924.

Microscopic examination of the purpuric spots in a case of meningococcus septicemia showed an inflammatory reaction of the subcutaneous stratum characterized especially by engorgement of the capillaries with leukocytes, capillary hemorrhage and numerous intracellular and extracellular meningococci. The epidermis was not involved. The author suggests that in some cases bacteriologic examination of the skin lesions may permit the making of an earlier diagnosis and treatment.

VONDERAHE, Cincinnati.

Society Transactions

NEW YORK NEUROLOGICAL SOCIETY

Four Hundred and Fourteenth Regular Meeting, Oct. 7, 1924

F. G. Zabriskie, M.D., *President, in the Chair*

Pathological Presentation: A Case of Cortical Tuberculoma with Operation. Dr. Lewis Stevenson.

A man, aged 20, one year ago complained of cramps in his left hand, so that he was unable to let go of a hammer he was using. This happened several times, but always passed off in a few moments. In April, 1923, ten days after the onset of symptoms, the patient suddenly lost conciousness, fell and stiffened. The next morning, he had another cramp in the left hand which was raised slightly, and then "it fell down;" there was no loss of consciousness. A few hours later, he again lost consciousness, fell and had twitching of his left arm and leg. After that, he had a number of convulsions, of jacksonian type, involving the left leg and left hand.

He had temporary paralysis of one arm and leg at the age of 2 years but he did not remember on which side. Otherwise the past and family histories were negative, except for some headache. In June, examination revealed: bilateral papilledema, definite weakness of the left leg with considerable weakness in plantar flexion at the ankle, normal abdominal reflexes, a positive Babinski sign on the left; spinal fluid, clear, no excess globulin; 23 lymphocytes per cubic millimeter; sugar, 40 mg. per hundred c.c.; Wassermann test, negative. A roentgenogram of the skull was negative. On July 22, a tumor was removed from the cortex of the right hemisphere in the region of the motor area for the leg. It resembled a conglomeration of peanuts as in peanut brittle and measured 1¼ by 1 by ¾ inches and was readily shelled out of its bed. On microscopic examination, many typical tubercles and giant cells were seen. No tubercle bacilli were found in the sections.

Immediately after operation, the patient was unable to move the left leg. Paralysis of the left arm also developed. There were no sensory disturbances before or after operation and no astereognosis. The patient still has a left-sided hemiplegia and occasional jacksonian fits involving the left side of the face and arm but apparently not the leg.

DISCUSSION

Dr. Sachs: In this case, symptoms of frontal lobe tumor were shown. The question was whether the symptoms were caused by a tuberculoma; also whether the growth should be treated by the roentgen-ray or radium. Tuberculomas are not rapidly fatal.

Dr. L. Pierce Clark: There was undoubtedly degeneration of the convolutions due to increased cranial pressure, and this produced transitory symptoms as well as discharge convulsions. To that extent it throws some light on the convulsive phenomena, but it could not in any sense be looked on as being of interpretive value in regard to essential epilepsy as such.

DR. S. P. GOODHART: As a rule, we should hesitate to operate because of the symptomatology. In this case, the finding of choked disk decided the diagnosis.

DR. KRAUS: What is the pathway of infection in this case? Why is it tuberculoma and not tuberculous meningitis? Tuberculoma involves the brain stem through vascular channels. Meningeal infections go through the lymphatics. Why was there this unusual finding of a tuberculous tumor of the cortex? Of course, cortical arteries are end arteries, as they are in the pons, but why did not the boy develop tuberculous meningitis?

DR. ELSBERG: Tuberculomas of the central nervous system are relatively rare, more frequent in the brain stem and cerebellum than in the cord, and is more often found in children. A smaller number occur in the hemispheres. I have seen two tuberculomas, both of which were hard, solid tumors, similar to the endotheliomas or meningiomas. In one patient, the tuberculous nature of the growth was in doubt until intensive pathologic studies had been made. In this patient, in spite of a negative Wassermann reaction, the tumor was at first called a gumma. At one time, Cushing made the statement that all patients with tuberculomas died in a short time from secondary tuberculous meningitis, and that he had never seen a patient who had been operated on recover permanently. At that time I was able to refer to two patients with tuberculoma of the hemispheres who recovered after operative interference, and both were well two years after the operation. Since that time, numerous other reports have been made of cases in which tuberculoma has been removed from the cranial or from the spinal cavity, without recurrence and without meningitis. It is now the generally accepted opinion that tuberculomas of the spinal cord are intramedullary; that when they occur in the hemispheres they are often cortical growths and often adherent to the dura. Tuberculous lesions in the brain stem cannot be subjected to surgical therapy. When found in the cerebellar hemispheres, secondary tuberculous meningitis is frequent, but not as frequent as it was once believed to be. I have recently operated in such a case at the Neurological Institute. Neither this patient nor others that I have operated on developed meningitis, so that meningitis is not a regular complication. When the tumor occurs in the spinal cord or over a cerebral hemisphere, it is easily removed. In the cerebellum, these growths often occur in the vermis, and this is the form that is least satisfactory from the standpoint of surgical treatment.

SOME PSYCHOLOGIC DATA REGARDING THE INTERPRETATION OF ESSENTIAL EPILEPSY. DR. L. PIERCE CLARK.

In previous papers, I have pointed out that essential epilepsy is characterized by no constant or enduring lesion of the nervous system. When a certain lesion is present, it is most likely to be a result of the general process of deterioration, constituting as it were a symptomatic pathology, and not in any specific sense a real cause of the disease. The great majority of researchers in this field freely admit that the most signal defect is a peculiar type of mental and physical deterioration. This is shown in a characteristic distortion and enlargement of the ego. I have presented data to show that these outstanding mental characteristics exist long before the disorder is recognized clinically. We see in this rigidity of the whole personality the dominance of egoistic traits that might be spoken of as a species of epileptic

behavior-pattern in its broadest sense. We find the most glaring faults bear a striking resemblance to those seen in persons with compulsion neurosis, and in those with praecox. In brief, the main traits of sadistic cruelty and hate, penuriousness, pedantry, and forms of piety and zealotry, are but the crudist forms of narcissism, supersensitiveness and emotional poverty.

Many have questioned whether the character defects alone, in their social discord, are sufficient to cause a later and enduring epileptic state. They cite the presence of similar personality faults almost as marked in other types, and yet epileptic reactions do not follow. Even so, it is the singular dominance and almost exclusive or pure culture of these character traits in the epileptic patient that make their presence of such dire consequence. Moreover, these dominant traits no longer rest on a trend of seemingly detached character faults, as formerly stated, but they have their origin in the deepest unconscious, and in the very organic substratum of the whole organism. In other words, the epileptic character is an outflow from a homosexual component which is not acceptably sublimated. While traces of this homosexual component may be shown in the epileptic physique, as might be expected, it is not so dominant, perhaps, as in the praecox. The mental characterizations are not directly of the homosexual type. How, then, is this component revealed? It may be said that it is present in the epileptic's peculiar type of narcissism. The narcissism of the epileptic is similar to that shown in the compulsion neuroses, but is of a much cruder pattern. What clinical evidence do we possess that the libido is inverted in the epileptic patient? Contrary to the popular belief, the epileptic patient is not sexually aggressive. As a class, they may practice onanism and may occasionally indulge in illicit relations during adolescence. Whether married or otherwise, their sexual life is not extraverted after early adult life. It usually ceases at 25 or 30 years of age. The main strength of the libido is turned back on the ego, building larger and larger the innate narcissism.

The seeming early asexuality of epileptic patients was attributed to the fact that they were closely interned and that sedatives were continually administered, but in the absence of both these factors, asexual phenomena were still in evidence.

Let us make a slight digression and see how the mechanism of sex inversion comes about. According to Freud, the two determining factors in the production of homosexuality are the love for the mother and the love for one's own body. These two components are invariably present and stand in the relation of contrast to each other, the narcissism being a result of the repression of the mother ideal; that is to say, the individual rids himself of the mother image as the love object by identifying himself with her, and substitutes his own person as the sexual or love object. Later, through an association of similarity, he extends his object to include other persons of his own sex. Thus we have the mother identification giving way to narcissism as the first step, the narcissism yielding homosexuality as the second.

Thus, if we may disregard the biologic inheritance, there is a distinct psychologic reason for homosexuality. In later life, such an individual completes his psychosexual union with someone who is more like himself and who better personifies himself; that is, his object love is unconsciously homosexual. If this unconscious trend is not sublimated into acceptable channels, he becomes a potential candidate for a neurosis.

The whole problem of the epileptic patient is not one of overt homosexuality nor one in which the fit is a libidinous discharge. It is far more

subtle. As a class, homosexuals are not recruited from the epileptic group. The early repression of the main homosexual trend probably never, or rarely, reaches consciousness. In point of fact, it only becomes dynamic in producing epileptic reactions when it is deeply repressed or strangled in the infantile unconscious.

The fit is a protective and regressive mechanism. It relieves the previous organismic tension and almost invariably either removes or makes amnesic the stress factor which wounded the unduly taxed narcissism. From a therapeutic point of view, it may practically be said that there exists but two main trends in the epileptic's libido—the heterosexual and the homosexual—and that no epileptic patient is without some factors of the heterosexual culture or behavior pattern. Providing natural outlets for development of these strong primary homosexual and narcissistic trends in self expression and personal ambitions is the easiest and immediate therapy. The more important principle is to encourage the development of the heterosexual trends of culture and behavior that lend to acceptable socialization. Unfortunately, ordinary psychanalysis is of little use. Most frequently, the heterosexual trend is so slight that these persons can make no proper transference. When it is established, it is based on such an infantile level as not to permit of adult socialization. Again, the narcissism so sensitizes the epileptic person that he practically becomes too deeply inhibited and is unable to awaken infantile memories. Though the verbal and even occasional visual memories are recovered, they are usually attended by no affect, and but little is accomplished, as is the case with the patient with praecox. Any modified analysis is extremely slow and tedious.

To summarize: I submit that the epileptic make-up has its roots in a narcissism which is based on a repressed or illy repressed homosexuality. It thus places the dynamic defect in the imperfect colligation of the two contrary trends in the sex instinct, and the dominance of the homosexuality and its incomplete repression are transformed or manifested by narcissism. The details of clinical data to support this tentative hypothesis will be presented at another time. Just what other factors are entailed in this imperfect development of the sex instinct—an epileptic in one instance, a paranoiac, a praecox, or a compulsion neurosis in other instances—is a problem requiring our most careful research.

DISCUSSION

Dr. Walter Koenigsberger, Berlin, Germany (by invitation): The speaker while elaborating a theory, gives us no data from which rational psychologic deductions can be drawn. One might reasonably ask for case material from the results of observation and treatment along the lines suggested. Before Dr. Clark can bring before us such a theory with emphasis, we may ask for psychanalytic examination and then the case reports, conclusions. Epilepsy has both physical and psychic phenomena. To ignore the probable and generally recognized rôle of physical and chemical agents and to see only symbolism and the rôle of the psyche, is asking much of credulity.

Dr. Smith Ely Jelliffe: I have listened to Dr. Clark attentively. I want to know what he means by "a new" psychologic interpretation. When he speaks of the desirability of viewing the patient with essential epilepsy as a constitutional personality type as wedged between the compulsion neurosis and the dementia praecox types, I can state that in 1915 Dr. White and I

put it there in our textbook. We thought that the closest alliances of the epileptic patient were with the compulsion neurosis and dementia praecox groups. Therefore I feel sympathetic to that disposal of the problem. Second, I find no fault with the general thesis of the homosexual libido fixation idea, but I do not see anything *new* in that. We have been thinking of the patient with essential epilepsy in that way for some time. In regard to the unconscious homosexual trends, we can concede that that generalization is valid. I would like Dr. Clark to emphasize the affective effort on the part of the individual to get his material over through the cardiovascular system. I think Dr. Clark is right in speaking of the stomach and gastro-intestinal system as being involved in homosexual identification factors. When he says that the fit is not "libidinal," I distinctly disagree and could cite numerous instances to show incestuous activities. The physiologic mechanism of the convulsion is centered largely on the vascular imbalance. I think that no one here who has had experience with either overt or unconscious expression of homosexual components will deny that the cardiovascular system bears the brunt of the repressed tension. I would like Dr. Clark to speak of that in detail and refer to Lewis' work on the cardiovascular system changes in dementia praecox and in paranoia, respectively.

I feel a certain amount of optimism in regard to the psychanalytic treatment of the patient with essential (I prefer the term "psychogenic") epilepsy. I do not know any other method that we can use, and I approve of the modification Dr. Clark has suggested in aiding the patient toward heterosexual transfer of his libido. I do not agree that the interpretation of the symbolism of dream analysis is not helpful in the therapy in epileptic cases. I think that it can be utilized to aid still further the affective realization toward the heterosexual goal. That the amnesia may be profound, everyone recognizes. That the transference is very infantile, is often also true. That type of infantile transference which is constantly shifting is one of the greatest difficulties in the therapeutic attack on the patient.

Dr. Philip R. Lehrman: I wish to point out the unity of mental phenomena in reference to reality. The symptoms of psychoneuroses, psychoses and essential (psychogenic) epilepsy indicate not only the intensity of the unconscious strivings, but also the necessity of the manner and degree of the withdrawal from reality. Thus, a partial withdrawal is seen in the psychoneuroses; a complete withdrawal is seen in the psychoses; and a sudden withdrawal is seen in epilepsy. The various mental symptoms are attempts at reparation and correspond to the need for a balance between the unconscious strivings and the interference of reality. The partial success of the sedative drug therapy in epilepsy can be accounted for when we bear in mind that these drugs partially accomplish what the symptoms attempt to do—they partially withdraw the patient from reality. Dr. Clark, however, directs his attention to the unconscious strivings which bring about the symptoms, and his studies are encouraging.

Prof. Kemball Young, of the University of Oregon (by invitation): I regard this paper as significant as dealing with epilepsy from the point of view of psychology rather than that of medicine. Dr. Clark has laid down certain grounds by which we can understand the whole development of personality from the unconscious, motor, vascular and muscular reactions. I do not think we can add anything to his clinical data. I believe that all the beginnings of the mechanism which underlie the epileptic attacks lie in the roots of the uncon-

scious and can never be recovered except by the study of the terms of the affect. When we understand compulsion and praecox cases we get at the muscular and vascular reactions of the child to the mother; and the study of the uterine state also throws some light on the subject. This type of personality is an egocentric one, and we can throw some psychologic light on it by study of the early reactions to show that there is a gradation through the crude form of epilepsy to the compulsion reactions and up to the reactions of the normal person.

DR. J. ROSETT: I have little to say regarding Dr. Clark's description of the character of the epileptic patient. That the average human being is in possession of more cunning and cruelty than any other animal is attested by the historical fact that of all animals man is the only one who actually delights in inflicting misery and pain on his fellows. Such being the case, the particular egocentricity of the epileptic patient is perhaps overemphasized.

Dr. Clark's assumption of deeply buried homosexual tendencies as a cause of epilepsy in those cases in which no pathologic lesion was found in the brain, appears to me as arbitrary. On the one hand, it is impossible to understand why a person with deeply buried homosexual tendencies would express them in the singular form of unconsciousness, rigidity and convulsions. On the other hand, the absence of a lesion in the brain of the patient with so-called essential epilepsy has never been proved. The most that can be said in such cases is that by the sectioning and staining methods at our disposal, we have been unable to discover it. As a matter of fact, how many such brains have really been carefully studied? The number of studies made of such brains by means of complete serial sections can be counted on the fingers. The available testimony regarding the absence of structural abnormality in the brains of patients with "essential" epilepsy is indeed too flimsy to be considered.

Sharp and Maclaire have made a discovery within the last year which appears to me of surpassing importance in this connection. They found blood in the cerebrospinal fluid of over 10 per cent. of new-born babies. Blood in the cerebrospinal fluid signifies an injury to the contents of the craniovertebral cavity. It means that a large percentage of human beings have sustained at birth an actual trauma of greater or less magnitude to their central nervous system. The fact that in a large majority of such cases the damage will have disappeared from view when the tissue is sectioned and stained years later speaks for the imperfection of our methods. A damage of structure, however, implies an abnormality of function.

In the light of these facts, the term "essential" epilepsy appears to me as highly irrational.

DR. OSNATO: I do not think that any one can quarrel with the statement that in a great many patients with epilepsy there may exist a highly important emotional factor. The interpretation may be what Dr. Clark says it is. Some of us who disagree may be pardoned for having concrete organic conceptions. During the development of physical emotional reactions, there are certain concrete physiologic biochemical factors which have been worked out by James, Lange, Cannon and Crile. There are certain chemical substances produced in emotional states which have been traced to the suprarenal glands, the thyroid, the liver, etc. Recently Cannon has isolated a protein histamin-like substance secreted from the liver. Whether one explains the emotional factors on a purely psychologic basis or whether one more satisfactorily explains them on

the basis I have mentioned, depends on the particular medical trend and point of view of the man making the explanation. The factor which produces the convulsion in epilepsy must work through the central nervous system. Whether this is produced in the central nervous system itself, I do not know. The reason we know so little about epilepsy, I say frankly, is because neurologists have had so much to do with it. The trouble may not be in the central nervous system at all, and I am of the opinion that it is not there primarily. In Dr. Stevenson's case, the tumor itself did not cause convulsions until it was able to cause vascular disturbance in the cortex. Dr. Jelliffe has referred to cardiovascular disturbance as a strong factor in causing the epileptic convulsion. In this boy, the seizures were caused by the tumor indirectly, perhaps disturbing cortical blood supply, while in the epileptic patient it may be caused by chemical disturbances resulting from improper metabolism. Many poisons cause convulsions when injected intravenously or painted on the cortex. It is not necesary even to have a cortex to have convulsions. Convulsions occur experimentally with the cortex ablated. Disturbances such as hemorrhages, trauma or neoplasm, at many levels from the cortex down to the medulla, may produce convulsions. Many mechanical or chemical factors may finally be found operating to produce the convulsion in epilepsy. In a small number of cases, the emotional factor may be dominant, indirectly causing the seizures by producing toxic substances capable of disturbing the blood supply in the brain (histamins, proteoses, etc.). Only in this way can I agree that a study of subconscious emotional factors is of any importance in persons with epilepsy.

Dr. E. G. Zabriskie: Essential epilepsy is decreasing day by day. The field is smaller. Each day, by improvement of examination methods, we find fewer cases. In regard to Dr. Clark's statements about the psychanalytic method, one of the main requisites of psychanalysis is not to construct an explanation, but absolute lack of prejudice, so that it would be possible to cite a series of cases in which the line of therapy would show simply the treatment of the deep complex as pointed out by Dr. Clark. I should like to ask whether experiments showed that general line of research method. I think there may be functional disturbances of the brain at first, but that would not make the changes which are seen later. Dr. Hauswitz of San Francisco thinks that the protective cushion surrounding the brain stem may collapse, so that sudden compression of the vascular system occurs with consequent stoppage of circulation, giving rise to sudden unconsciousness; thus an instantaneous deprivation of blood to the brain itself may cause the convulsion. I stayed at the Craig Colony to make observation on this subject. I thought that if the unconsciousness could be produced by ligature of the basilar artery, with collapse of the watery cushion below the brain stem, the brain stem might become impacted against the bone at the base of the skull. The aura is said to make a sudden darkening before the eyes of the patient, which might be produced by the closing off of blood coming to the eyes; 50 per cent. of patients speak of this darkening as reported in the textbooks, but at the Craig Colony I did not find one patient with this experience. I feel that functional disorder may exist in the brain without making visible changes of structure.

Dr. Bernard Sachs: The trouble with this subject is the same as with psychanalysis. Starting with presumptions which have not been proved, the author builds up a structure purely fantastic. Dr. Clark's personal experience

is very large, but I do not think that he will be able to prove clinically that the vast majority of patients with epilepsy are homosexual, that they cannot be tempted into the heterosexual field, or that their libido is invariably improper; the patient with epilepsy behaves as the average human being behaves. Fortunately, Wohlgen, in his book, has knocked the bottom out of these theories which we hear paraded around as though they were proved. I have tried to study these questions from the point of view of one interested in philosophy and psychology. It is difficult to follow the exposition of theories that are obscured by a ridiculous verbiage. As a matter of fact, there is no logical basis for the symbolism of psychanalysis. Personally, I should be glad if an entire psychologic basis for epilepsy were found, but I am more in sympathy with Dr. Rosett's views than with Dr. Clark's. I do not believe there is any epilepsy without cerebral disturbance, either due to structural changes in the brain or to vascular instability. I believe the emotional instability and deterioration in epilepsy are largely to be ascribed to the morbid process causing the epilepsy. Formerly, we thought the use of drugs produced the mental deterioration. Now we give less drugs, and we see less deterioration. There may be a disordered personality, but do not call every man who takes thought for his body a victim of narcissism. That is the latest catchword.

Dr. L. Pierce Clark: I wish to apologize for the word "new"—the material is not new, but its application is. I have been working on the subject of essential epilepsy for nearly as long a time as Dr. Sachs. I know the literature that covers the subject as regards etiopathology, and yet I am constrained to come to a different conclusion than Dr. Sachs. I believe the ultimate explanation of essential epilepsy will be found in a defect in the functioning of the organism as a whole, and not in any part reaction or resultant of alteration of bodily functions such as gastro-intestinal and vasomotor disorders, muscular deficiencies and general metabolic faults. One might ask, how are we to divide the strictly so-called functional from structural processes? Any attempt at such a schematic conception is worn out and is being rapidly discarded. Anything less than a dynamic explanation of the functioning of the organism as a whole must be discarded, and partial reactions can never explain essential epilepsy. In the psychoneuroses of the compulsion type and in essential epilepsy we must think of the individual as a living, functioning organism operating in its entirety through partial reactions but not because of them. Blood or serologic reactions in their turn have no significance except as they participate in the total reaction. For instance, paranoia is not a result of a lesion of the brain, nor is bad temper; but these behavioristic reactions are fused into the individual as a whole. Even neurology and psychology fail to cover the whole field of the psychoneuroses. We must take into account the general concept of the individual as well as his relationships to environment. We must correlate the mind, body and environmental relationships to gain any comprehension of the totality of normal as well as of psychotic behavior. Subjective as well as objective data when taken together constitute as good clinical evidence as any so-called neurologic science.

Neurology in Paris, 1923-1924. Dr. Walter M. Kraus.

Dr. Kraus presented a paper describing the organization of neurology in Paris as well as the activities of various men there during 1923 and 1924.

BOSTON SOCIETY OF PSYCHIATRY AND NEUROLOGY

Regular Meeting, Nov. 20, 1924

C. MACFIE CAMPBELL, M.D., *President, in the Chair*

AN UNUSUAL CASE OF EPIDEMIC ENCEPHALITIS. DR. HARVEY B. SANBORN.

A woman, aged 36, married, whose family history was irrelevant and whose personal past history was negative except that she had been a "high liver" and perhaps overindulged somewhat in alcoholic beverages, was operated on, Nov. 26, 1923, for left mastoiditis, and for a few days before and following the operation showed paralysis of the external rectus muscle on the side of the mastoid. By Jan. 1, 1924, the ear and the mastoid wound were well healed, and she seemed to be making a good recovery. On January 11, the patient again became acutely ill with fever, intense headache, herpetic eruption on the right side of the face and severe pain at the site of the eruption. On January 15, her temperature was 100 to 101 F.; she showed slight drooping of the right upper lid and some dilatation of the right pupil; both knee reflexes were diminished. The diagnosis made on this date was herpes zoster following mastoid infection. On January 23, she showed complete right ophthalmoplegia without other evidence of cranial nerve involvement, and complete loss of the knee reflexes and the Achilles reflex. Fever persisted. The spinal fluid showed 23 cells, all lymphocytes, and increased globulin. The Wassermann reaction on the blood and spinal fluid was negative. Examination of the eyegrounds was negative. There had been no lethargy. The patient was depressed and irritable. A diagnosis of probable epidemic encephalitis was made and later concurred in by Drs. E. W. Taylor and Channing Frothingham, although we all felt some doubt.

The fever persisted intermittently for about four weeks; the headache and facial pain gradually disappeared, and after several weeks, power in the eye muscles began to return. The knee reflexes returned after about two weeks. Some time in May, or about the time she began to get out and about and again partake of alcoholic drinks, she began to have excessive thirst and polyuria, and this has persisted. The findings in October by Dr. Frothingham were typically those of diabetes insipidus, and the symptoms are now being held in check by intramuscular injections of pituitary extract twice daily. There is still slight weakness of the extra-ocular muscles, and the pupil remains widely dilated.

DISCUSSION

DR. E. W. TAYLOR: I do not yet feel altogether sure that encephalitis is the correct diagnosis, but in the absence of any other, by exclusion, that is the safest. The herpes and the presence of previous definite infection of the mastoid complicated the situation materially, as did also the definite alcoholic history. She was irritable, and her temper was most extraordinary; whether or not that was habitual, I do not know. I presume the diagnosis of encephalitis is the most probable one, but there was scarcely a feature of it which seemed to me in any sense characteristic. Complete unilateral ophthalmoplegia must be exceedingly rare.

DR. CHANNING FROTHINGHAM: Did this patient ever have acute otitis media, and did she ever have a mastoid infection? I do not feel sure that she ever had either, and I think it conceivable that the symptoms of acute

middle ear infection and the mastoiditis may have been part of the encephalitis. If we could rule out the acute middle ear infection and mastoiditis, would that make a diagnosis of encephalitis any easier? The trouble in the first place was in the left ear, then in the left mastoid region, and finally swung over to the right side of the face. The next question is: What is the end-result? Has the patient at the present time diabetes insipidus resulting from the encephalitis, or is she just slowly recovering from the paralysis, the prostration, diarrhea and fever? Are her present symptoms the result of her habits? She insists that she developed a tremendous thirst before she began to drink alcohol again after her illness. Although she drinks alcohol, perhaps more than she ought to, it does not seem to me that she drinks enough to account for diabetes insipidus. I had the opportunity to observe her in an environment where she was on a fixed level of life, receiving just a little alcohol and following a regular routine of diet and physical exercise. Following that routine, she drank practically 10 liters of water a day, and the daily output of urine was 10 liters. When she was given pituitary extract subcutaneously on the same routine, the output of urine dropped to 5 liters and then to 3 liters a day. After stopping the injections and giving her the pituitary extract by the nasal spraying method, the output of urine promptly went up again, at first to 6 and then to 8 liters a day. The polyuria and polydipsia were markedly influenced by the pituitary extract, as would happen in diabetes insipidus. Then the question arises: Was this a result of encephalitis, or has she, perhaps, some disease of the pituitary gland or central nervous system which has given her all these various symptoms in her eye and ear, and now diabetes insipidus? I should like to ask Dr. Sanborn whether diabetes insipidus is a common sequel of encephalitis. The patient's basal metabolism is normal, her skull plate is normal, and her eyegrounds are normal at the present time.

DR. MABEL ORDWAY: In 1918, Dr. Beverly Tucker discussed the involvement of the pituitary gland in encephalitis.

DR. PERCIVAL BAILEY: So far as I know, no one has shown definitely that the causal lesion in diabetes insipidus lies in the hypophysis. The clinical and experimental evidence points rather to a lesion of the hypothalamus. In this patient, the diabetes insipidus might well have been due to such a lesion, since the encephalitic virus is markedly neurotropic. Diabetes insipidus is not a rare sequel of epidemic encephalitis, and in all cases investigated no lesion has been found in the hypophysis.

DR. J. W. COURTNEY: Foster Kennedy has reported an extraordinary case of diabetes insipidus following encephalitis; it must be recognized as one of its sequels.

DR. C. MACFIE CAMPBELL: Diabetes insipidus as a result of encephalitis has been observed at the Boston Psychopathic Hospital.

DR. SANBORN: Whether or not the patient's irritability was habitual, I do not know, but after talking with the family physician I should judge it might be. It might well have been accentuated by the illness, but I think that latent irritability was there all the time. I have talked with the aural surgeons who had charge of the case, and they seem to feel that the operation showed definite signs of mastoid infection. We find in a majority of the cases of encephalitis a history of preceding infection of the respiratory tract, and what the relationship between the two conditions is, we are not sure. In this particular case, we might conjecture that the mastoid infection simply served to lower her

resistance, or there might be a more intimate relationship.. On the other hand, if we wish to theorize and say that possibly the mastoid pain and the transient external rectus paralysis were not due to actual mastoid infection but the earliest manifestation of an epidemic encephalitis, then such a clinical picture is not out of harmony with other known cases of encephalitis in which there has been a period of remission between different manifestations of the disease. As to diabetes insipidus being a sequel of encephalitis, I was under the impression that there have been a number of such cases reported.

COMBINED VENTRICULAR AND LUMBAR PUNCTURE IN THE DIAGNOSIS OF BRAIN TUMOR. DR. FRANK FREMONT-SMITH and DR. JOHN S. HODGSON.

DR. FRANK FREMONT-SMITH: In certain patients, complete neurologic study is not possible; in others, the result of examination may still leave in doubt the localization of an intracranial tumor. In such cases, we suggest the use of combined ventricular and lumbar puncture as an aid to localization. Our method is based on Ayer's work, and follows as a logical sequence to it.

The lateral ventricle is punctured in the usual way, and an initial pressure reading is obtained; then enough fluid is withdrawn to lower the pressure to within normal limits. Lumbar puncture is now performed, and simultaneous pressure studies are carried out in the same manner as described by Ayer (ARCH. NEUROL. & PSYCHIAT. **7**:38-50 [Jan.] 1922).

Normally, there is free communication between cistern and lumbar subarachnoid spaces. A sufficient number of ventricle punctures combined with lumbar punctures or with puncture of the cisterna magna have been made by Mixter and Ayer in cases of acute meningitis, and by Solomon and others in cerebrospinal syphilis, to demonstrate that the same free communication obtains normally between ventricle and lumbar subarachnoid space. Thus with a patient in a lateral position manometers attached to lumbar and ventricle needles will register equal pressures. Withdrawal of fluid at either locus will cause a simultaneous fall in each manometer. Jugular compression will give prompt, simultaneous and equal rise in pressure in both ventricular and lumbar manometer, and on release of jugular compression, both fluid levels will promptly fall to the original level or a little higher. Pulse and respiratory oscillations will be noted in each manometer, and there will normally be a slightly greater amplitude in the ventricular manometer. Any change in pressure within the fluid spaces will be registered immediately and equally in the two manometers. These are the normal relationships, wholly similar to those obtained by lumbar and cistern punctures.

Complete spinal subarachnoid block manifests itself by abnormal pressure relationship between lumbar sac and cisterna magna. Changing the pressure in one locus will fail to register by a corresponding change in the other. Of the various tests, the most important is the response to jugular compression. In complete block, no rise will obtain in the lumbar manometer when the jugular veins are compressed, while normal rise and fall will occur in the cisterna magna. In many early cases, Ayer has demonstrated partial dynamic block. This is shown by a relatively slow and slight rise in the lumbar manometer on jugular compression, with delayed fall on jugular compression release.

A tumor or other lesion causing block between ventricular and lumbar fluid should manifest itself in the same way. It must be borne in mind, however, that complete obstruction to the outflow of cerebrospinal fluid from the ventricular system is compatible with life for only a short time, provided the

cranial sutures are ossified. When the outflow is partially restricted, the ever rising intraventricular pressure tends to keep a partial communication open. Complete block, then, is not to be expected and has not been our finding.

Ayer made use of chemical as well as hydrodynamic differences in the two fluids, and emphasized the fact that the increase in protein in the fluid below a partial spinal subarachnoid block may occur earlier than demonstrable differences in hydrodynamic relations. Protein is also increased in the fluid above a spinal subarachnoid block, but this increase is relatively slight. The increase in protein is also found below certain brain tumors, and may be striking when there is little or no evidence of dynamic block. There is general agreement that normally the ventricle fluid contains a little less than half the protein found in the lumbar fluid. Cestan (Cestan, Riser and Laborde: Rév. neurol., April, 1923, No. 4, p. 353) gives 10 mg. per hundred cubic centimeters as a normal value in the ventricle, and 30 mg. in the lumbar. In our laboratories,

Combined Lumbar and Ventricular Puncture

		Protein	
		Lumbar	Ventricular
1. Cerebellar glioma	Partial block	61	18 left 35 right*
2. Cerebellar glioma	Partial block	64	24
3. Cerebellar arachnoid cyst	Partial block	66	23
4. Fourth ventricle tumor	Partial block	38	7
5. Fourth ventricle tumor	Partial block	37	12
6. Cerebellar-pontile angle tumor... Acoustic neuroma	No block	400	13 left 13 right
7. Cerebellar-pontile angle tumor... Acoustic neuroma	Partial block	267	8
8. Cerebellar-pontile angle tumor... Endothelioma	Partial block	181	15
9. Parietal arachnoid cyst	No block	44	16
10. Temporofrontal arachnoid cyst	No block	55	342*
11. Glioma: left motor cortex	No block	27	33*
12. Glioma: left frontal lobe	No block	45	20
13. Left frontal tumor	No block	40	24*
14. Glioma: left motor cortex	No block	138	103
15. Tetratoma: third ventricle	Not simultaneous	200	129

* Red blood cells in fluid.

we have found normal ventricular protein to range between 5 and 20 mg.; while the lumbar values lie between 12 and 40. In no case have we found the ventricular protein to be greater than half the lumbar when normal relations exist. When block between lumbar and ventricular subarachnoid space is present, we have found the ventricle fluid protein normal and the lumbar fluid varying from normal to 400 mg. per hundred cubic centimeters.

As would be expected, it is the tumors lying below the tentorium which most commonly produce dynamic block. We have had no experience with the more rare midbrain tumors or those in the region of the pineal gland.

The accompanying table summarizes findings in fourteen cases of combined puncture. Case 15 is added because of high ventricular protein. The tumor projected into the lateral ventricles.

In most of these cases, the diagnosis was made correctly without the aid of combined puncture. In no case have the results of this procedure been contrary to the correct localization. In three cases, combined puncture has been of definite aid in diagnosis.

Summary.—Combined ventricular and lumbar punctures in fourteen cases have given the following results:

1. Partial dynamic block in subtentorial tumors or cysts—seven out of eight cases.

2. Increased protein in lumbar fluid in subtentorial lesions—six out of eight cases (two fourth ventricle tumors showed little if any increase).

3. Marked increase of protein in lumbar fluid in cerebello-pontile angle tumors—three cases (two acoustic neuroma, one endothelioma).

4. No dynamic block in tumors or cysts of the cerebrum (intraventricular tumors excepted) six cases.

5. Slight or no increase in protein in lumbar or ventricular fluid in cerebral tumors (intraventricular tumors excepted).

Finally, we wish to make clear that this is a preliminary report on a procedure which is still in the experimental stage. We do not yet offer it as of proved value.

DR. JOHN S. HODGSON: Dandy, in cases of brain tumor in coma, in which none of the usual methods of localization were of value, has performed bilateral ventricular puncture. He aspirates ventricular contents, and injects indigo carmine into one ventricle, recovering the solution either from the other ventricle or at operation from the cisterna magna. He has suggested the possibility of recovering the dye from the lumbar region.

So far as we are aware, combined ventricular and lumbar puncture, and the hydrodynamic and chemical study of the two fluids as an aid in the localization of brain tumor has not hitherto been reported. We believe that this method is helpful wherever localization is impossible or doubtful by other means. This includes cases of coma. The procedure enables us to obtain the lumbar fluid and with less danger of medullary injury than in lumbar puncture alone. In cases with block, it is essential that only a minimum amount of lumbar fluid be withdrawn. Concerning the technic, the patient is placed on the side as for simple lumbar puncture. Local anesthesia is generally used. The ventricular needle should be of the same bore as the lumbar and has a three way connection into which fits a manometer. The posterior horn of the vestibule is the site most commonly chosen for the tap. If both lateral ventricles are tapped, block between them can be determined. We have had no case in which this existed. Ventricular puncture precedes lumbar puncture in all cases, and the ventricular pressure is lowered to normal limits before lumbar puncture is performed in order to avoid the danger of medullary injury. Immediately following ventricular puncture lumbar puncture is performed, and simultaneous manometric pressure readings are made in ventricular and lumbar regions. The rate and amplitude of pulse and respiratory oscillations in the two manometers are compared with and without jugular compression or cough. Response to jugular compression in the two loci is most important as indicating the presence or absence of dynamic block. Fluid is then removed, first from the ventricle and then from the lumbar region, the effect on the ventricular and lumbar pressure levels noted, and jugular compression is repeated. The final pressures in the ventricles and lumbar region must be left equal and normal.

We have injected air into the ventricles in the majority of our cases. In the absence of block, the injection may follow combined puncture; in the presence of block, the injection should be made later. Cases of block have

to be carefully watched following either combined puncture or pneumoventriculography for early signs of respiratory failure. Should these occur, immediate relief may be obtained by ventricular tap. In one of our early cases in which partial block was present, air was injected at the end of the procedure. Twelve hours later, the patient suddenly died. Necropsy showed a large cerebellar glioma with pressure cone.

We are reporting a small series of cases in which we have found combined ventricular and lumbar puncture a valuable adjunct in the localization of certain brain tumors. The method is not intended to replace the usual means of diagnosis. We believe that results have been sufficiently encouraging to justify its continued use.

DISCUSSION

Dr. J. B. Ayer: Any mechanical procedure which sheds light on a doubtful diagnosis should be welcomed. It is likely that this procedure falls into this category. The technic is logical, and if we admit that ventricular puncture is a procedure which may reasonably be employed frequently, as has been vouched for in a recent paper on meningitis, then we should not hesitate to employ this method in this important group of cases. While, of course, the results obtained in this small series should not be taken as final, it would seem as if block could be demonstrated almost constantly in tumors below the tentorium, whereas it seems quite as certain that tumors above the tentorium do not produce block. This is exactly what one would expect, and it is reasonable that these workers have obtained such results. The findings in cord tumor are similar but more striking. It has been suggested by a number of workers that there are characteristic findings in the lumbar fluid in cases of brain tumor. Stress has been laid in particular on the colloidal gold changes, but increased protein has also been mentioned as a characteristic finding. In our laboratory, these two abnormalities have been encountered, but no constant finding has been obtained. Certainly, no localizing sign has been obtained by examination of the lumbar fluid in our clinic. Therefore, we welcome this new procedure as of hopeful aid. As a by-product of this work, we may call attention to the fact, insisted on by many, that lumbar puncture in brain tumors in the cerebellar fossa is dangerous. This fact discourages us from examining the fluid where such a possibility exists, although we frequently are most anxious to know what the fluid in such a case would show. It is possible that the preliminary withdrawal of the fluid from the ventricle will allow us safely to perform lumbar puncture in these cases, although it must be admitted that even this technic is not free from danger.

Dr. Bailey: Drs. Putnam and Sterling have made two such observations in our clinic on two patients in whose cases the diagnosis was in doubt. We all know of many cases of brain tumor which we are unable to localize, and any procedure which will aid in the localization in these cases should be given every possible consideration.

Dr. W. J. Mixter: I have watched this procedure with a great deal of interest, and from the surgical angle it seems to me that it is something that gives every promise of being a distinct help. One of the greatest difficulties I have in the localization of brain tumors is the differentiation between frontal lobe and cerebellar lesions. This has come up at various times in the past. This procedure may give us the key to the differentiation between frontal and cerebellar tumors which are otherwise not open to localization.

SOME FURTHER CASES OF EPENDYMOMA. DR. PERCIVAL BAILEY.

The clinical histories of seven patients with brain tumor were presented. Examination of the specimen removed at operation or at necropsy showed the tumors to be ependymomas. Three were in the region of the fourth ventricle, three in the cerebrum and one in the spinal canal. In all of them, the typical granules known as blepharoplasts were clearly visible in the cells.

News and Comment

ARCHIVES OF OTOLARYNGOLOGY

We take pleasure in announcing the appearance of a sister publication. Volume 1, Number 1, of the *Archives of Otolaryngology*, which is to be published monthly by the American Medical Association, has just appeared. The members of the Editorial Board are: George E. Sharmbaugh, Isidor Friesner, Chevalier Jackson, Eugene A. Crockett, Robert C. Lynch and Greenfield Sluder. If this first number is any indication of the quality of the new journal, it doubtless will quickly assume the leadership of all publications on otolaryngology in this country.

Book Reviews

LEITFADEN DER ELEKTRODIAGNOSTIK UND ELEKTROTHERAPIE. VON PROF. DR. TOBY COHN, 7te Auflage. Price, 7.20 marks, unbound; 8.10 marks, bound. Pp. 232. Berlin: S. Karger.

This manual is a fairly comprehensive survey of the field of electrodiagnosis and electrotherapy, designed for students and practicing physicians. The fact that it has passed through seven editions since its first appearance in 1898 would indicate that it has served a useful purpose. The first half of the book deals with electrodiagnosis. The fundamentals of physics and physiology are briefly and adequately explained. The various pathologic reactions are given with commendable clarity and simplicity, and are complete enough for all clinical needs. The only new feature in this section deals with chronaxie; it is sketchily handled, as the author feels that it has not yet proved itself of sufficient practical value in diagnosis and prognosis to warrant detailed discussion. There are two interesting pages in this section devoted to a discussion of electrical examination in relation to diagnosis, prognosis and treatment of gunshot wounds as observed in the war. A partial reaction of degeneration means that the outlook is good, and calls for conservative treatment (physiotherapy). A complete reaction of degeneration means that the lesion is severe, but tells nothing of the anatomic continuity of the nerve. Many such patients will recover under conservative treatment. It is only when the galvanic irritability drops from week to week, along with a persistence of slow contraction wave and displacement of the point of stimulation, that the prognosis is bad and operation unconditionally advised, "though even in these cases operation may show that the nerve is relatively intact and capable of spontaneous recovery." Between these two extremes, and this includes the mass of cases, the electrical examination can give only a little help, and the decision as between surgery and physiotherapy must be decided on other grounds—largely on the leanings of the physician. The author's point of view is decidedly conservative. He favors electromechanotherapy carried out for four or five months with careful rechecking of the findings from time to time. He deplores the attitude of those who consider an unnecessary operation less risky than a prolonged wait. In view of the fact that it is frequently impossible to recognize anatomic division of a nerve by any means at our disposal, it would seem that early exploration has much more in its favor than the author is willing to concede.

The second half of the book deals with electrotherapy, a field which has always been relatively neglected in this country, and which, in spite of the known accomplishments of the reconstruction hospitals during the war, is still neglected. The new edition of this work is especially welcome on that account. And yet, when one looks over the indications for treatment as laid down and scans the lists of diseases for which the various forms of electrotherapy are advised, he is amazed. It suggests much that we are accustomed to associate with the extra-medical fraternity of healers. Some of the suggestions, it is true, he mentions only to condemn. But there are recommendations for treatment by galvanism and faradism of such a variety of conditions that one cannot but feel that any benefit must depend on the one common factor of suggestion. On the other side

of the scale, we should unquestionably give more attention to the electrical treatment of peripheral nerve lesions and anterior poliomyelitis. Nothing will replace the galvanic type current in producing contractions in paralyzed muscles.

Electric baths are warmly recommended in a variety of conditions, mainly for their tonic effect. Franklinization and the various forms of high frequency current are discussed, with especial emphasis on the use of diathermy. Diathermy is an effective method of producing deep-seated hyperemia. Its indications can be deduced from its physical effects.

The paper and printing are excellent. The diagrams, charts and illustrations are good; those giving the motor nerve points are exceptionally so.

PATHOLOGIC ANATOMY OF THE SENILE PSYCHOSES. By PROF. ARRIGO FRIGERIO. Pp. 158. Pesaro, Cav. G. Federici, 1923.

The phenomenon of old age and the various psychopathologic problems connected with it occupies a place of no little importance in the field of psychiatry. In the study of the so-called senile psychoses a great mass of anatomopathologic observations have been collected recently, but we are still far from a consensus of opinion as concerns the pathogenetic interpretation of some of the most important symptoms, and we are unable to make a definite nosographic classification. It is conceded by the author of this book, who admits, in spite of all his researches, that we still have to rely on clinical observations rather than on the histopathologic findings for the diagnosis of senile dementia. The book has two parts, one being a critical review of the facts already known of the pathology of the senile psychoses and their symptomatologic relationship, giving the opinions of the different psychiatric schools on the subject, and the other dealing with his personal anatomoclinical studies of a certain number of cases, whose necropsies are reported in full.

Such subjects as vascular alterations (hyalin, colloid, calcareous degeneration), alterations of the elements of the nervous substance (Nissl body alterations, granulovacuolar degeneration, alterations of the neurofibrillar reticulum of the nervous fibers and neuroglia), complicated formations, such as senile plaques, amyloid bodies, lacunae of disintegration, on whose formation there is no accord, are discussed thoroughly in the light of modern researches and compared with the author's findings. The visceral alterations, particularly renal, hepatic and thyroid, are dealt with in a special chapter.

From the original researches of the author, it appears that we possess definite ideas as to the signs of physiologic involution of the nervous system, but not on the characteristic elementary alterations of the senile psychoses. Senile dementia as the consequence of a physiologic involution of extreme degree must be considered an exception. Alzheimer's classification of senile dementia and arteriosclerotic dementia remains unchanged. Both forms are prevailing in old age, but may exist also in early periods. These two types are based on a common autotoxic origin, but are differentiated by anatomopathologic manifestations. They may be combined in the same patient; both may be complicated by acute episodes, and by the presence of factors of congenital lability of some systems of neurons. This fact accounts for the various syndromes which are often considered as distinct forms, but which may exist independent of age and often in evident relationship with known etiologic factors, such as alcoholism for the presbyophrenic syndrome. The senile plaques do not represent a sufficiently real pathologic process to be responsible for the psychic symptoms. They are found in normal subjects and must be considered related essentially to age. As to the neurofibril lesions of Alzheimer,

the author defers final judgment until more accurate observations in many more cases have been collected. He thinks, however, in spite of the recent attempt to minimize their value, that such serious cellular lesions, if disseminated, cannot exist without consequence to the intellectual activities. Alzheimer's syndrome should not be separated from the group of senile dementias. Owing to its appearance also in young subjects (in order to avoid confusion), it should not be called senile. As to the other forms included among the senile and presenile psychoses, the anatomic pathologic findings do not warrant their approach to any of the two groups; they cannot be considered to be in direct relationship with old age; on the contrary, some histopathologic findings suggest their approach to the other forms.

An up-to-date bibliography and fifteen photomicrographic tables close the book. The author shows a solid grasp on the questions he deals with, and a vast range of information. This book represents a real contribution to psychiatry, and it can be recommended to those who are in search of recent data on senile psychoses.

INSANITY AND THE CRIMINAL LAW. By WILLIAM A. WHITE, M.D. Price, $2.50. Pp. 284. The Macmillan Company, 1923.

White's treatise is an unusually interesting exposition of the relationship between the law and the expert psychiatric witness. It is particularly timely, since at the present time the general estimate of the alienist in his expert capacity is far from flattering. White clearly shows how helpless is the medical witness once he exposes himself to the innumerable technicalities of the legal process. The weakness of the position in which law places him is chiefly due to the fact that he cannot escape partisanship; in other words, he must be either a witness for the prosecution or for the defense. Even should he desire to do so, he cannot hope to be permitted to deliver a medical opinion with the necessary honest qualifications and reservations, such as, for instance, he might render after the examination of a mentally sick person in private practice. The personal and professional character of the great majority of the alienists who appear in court is such that no one should impugn their motives or doubt their sincerity. However, they continue, perhaps often unavoidably, to help perpetuate a system which serves little or no constructive purpose and does not materially assist the administration of justice. From the standpoint of our own specialty (psychiatry), this is all the more regrettable since we have so much that is valuable to contribute to the development of criminology. It is often public opinion that the alienist sells himself for a large fee. Frequently public opinion is wrong, and undeniably it is erroneous in this instance; and yet it is clearly worth while to make it more difficult for the public to arrive at such a conclusion.

The author embodies under "Legal Suggestions for Betterment" the crystallization of the discussion of a Committee of the American Institute of Criminal Law and Criminology. They represent a notable advance in medical jurisprudence and are quoted in full. Many excellent suggestions are made; especially noteworthy is the summoning of the panel of experts by the judge, who shall individually read at the time of the trial a report of their findings. White, himself, would have the jury pass solely on the question as to whether the defendant committed an antisocial act and not on his guilt or innocence. However, as Professor Edwin R. Keedy pointed out, such restriction would be unconstitutional since "The constitution guarantees the right of trial by jury. This guarantee means more than that twelve men shall sit together in the

court room during a defendant's trial. It means that the defendant has a right to have the necessary elements of his guilt passed upon by the jury. According to the law, criminal intent is a necessary requisite of crime. Consequently the jury which decided whether the criminal act was committed must determine whether the criminal intent was present or absent." The reviewer does not call attention to the potential unconstitutionality as a valid objection to the merit of the plan but merely from the standpoint of expediency. Probably White had this same question of constitutional right in mind when in Section I of the Expert Testimony Bill it is stated that "Such calling of witnesses by the court shall not preclude the prosecution or defense from calling other expert witnesses at the trial."

Throughout the book there are numerous advanced concepts expressed which, if put into practice, would go a long way toward rescuing criminal law from its present unenviable rôle as the pariah among the legal specialties. For instance: White argues, why should not judges specialize in the study of certain criminal tendencies, and young lawyers, on their graduation, spend some time as interns in prisons, observing and studying the criminal? The medical intern secures his most valuable experience in the general hospital, living and working among the sick. This method makes far more efficient physicians; such a method would result in more able and better trained lawyers, and criminal law would eventually be numbered not among the lowest but as the highest and noblest of legal branches. Certainly the study of men, especially sick men, as are a large proportion of criminals, is in the last analysis more constructive socially, morally and economically than a determination of the rights of corporations can ever hope to be.

White's theory of crime and punishment follows along the lines of analytic psychology. The crime is behavior directed against the interests of the herd, and guilt and punishment as determined by the jury (representing the herd) and administered by society is the expression and survival of archaic vengeance in sublimated form. Of course, there is much basic truth in this statement. However, it is probable that the author is rather too meager in his idea of the progress which he conceives society has made away from the law of private revenge or lex talionis. One may freely grant that not infrequently in the verdicts of juries may be read a more or less direct emotional response, which may transgress the rules of the law and disregard the weight of evidence. So, too, when for instance the primitive urge of the mob sweeps aside the recorded opinion of the law or protests against its delay by exacting a bloody revenge. However, there is another side of the picture which must also be placed on view. There is no doubt that the very order and method of legal procedure often prevents primitive expressions either for or against the criminal. In other words, the "rules of the game" or the body of legal doctrine, faulty though it may be, is still too authoritative to be cast aside whenever emotional stirring prompts. Certainly, the tremendous difficulty which prosecutors experience in attempting to secure a verdict and execution for murder in the United States is not convincing evidence in favor of the argument that at the present time punishment is still the active even though sublimated expression of vengeance that an individual or a tribe visited on the person of the transgressor in the past. While it no doubt still operates from time to time, yet it seems likely that generally it is more symbolic than actual. Perhaps, the crux of the matter lies in the concept of the law. White believes it is transient and unchanging, springing from and reflecting the customs, habits, usages and thoughts of society; in other words, the law is

unwritten and unconscious, while statutes are merely supplementary. This is essentially the point of view of Carter, but from this attitude Professor Keedy and other eminent legal minds dissent. White sees in the crime the concrete fruition of what has gone before. Such deterministic psychology is without doubt valid, and certainly it would have the support of every psychiatrist. To a certain extent, this is true not only for the criminal act but for every kind and quality of behavior. On the other hand, the practical application of this philosophy, its carrying over for instance into law, will have to be accomplished with the greatest caution and much reservation, since its sudden unconditioned application would strike at the very roots of social evolution and produce chaos. From his speculations in this direction, the author draws the extremely valuable lesson of studying the criminal as an individual and not as a type, which should eventually lead to a salvaging of some of the tremendous waste of energy which accompanies our present mode of administering punishment to the criminal.

"Insanity and the Criminal Law" is worth careful study and consideration. Its theory is always interesting even when one is not in agreement with certain of the hypotheses. The many practical suggestions it contains are in themselves sufficient to justify its writing.

GROUP PSYCHOLOGY AND THE ANALYSIS OF THE EGO. SIGMUND FREUD, M.D., LL.D. Trans. by JAMES STRACHEY. Pp. 127. Price, $2.00. New York: Boni & Liveright, 1924.

Quotations from Le Bon and McDougall sum up the psychologic behavior of a simple unorganized group: excessively emotional, markedly reduced intellectually, easily swayed and led, acting at times like an unruly child and at others like a passionate savage. For Freud, the problem of raising collective mental life to a higher level would consist in equipping the group with the attributes of the individual, which were extinguished in him by the formation of the group. After calling attention to the extended use and loose meaning of "suggestion" and to the efforts to fix correctly its conventional use, he temporarily evades the riddle and inserts the concept libido, i. e., "the energy of those instincts which have to do with all that may be comprised under the word love." Sexual love, love for parents and children, friendship and altruism, devotion to concrete objects and to abstract ideas—all these tendencies are an expression of the same instinctive activities. Love and libido, therefore, as used by Freud are understood to have this wider sense.

He now advances an entirely new supposition that love relationships or emotional ties, as well as suggestion, constitute the essence of the group mind. Of the many types and grades of groups, he calls attention to the distinction between those with leaders and those without, mildly reproaching the authorities for not having sufficiently appreciated the importance of the leader. He cites the highly organized, lasting and artificial groups—churches and armies. No matter how different in many respects, the same illusion holds good of there being a head—in the church, Christ; in an army, its commander-in-chief—who loves all the individuals in the group with an equal love. Christ is the father surrogate, and all the demands that are made on the individual are derived from his love. The tie which unites each individual with Christ is also the cause of the tie which unites them with one another. The commander-in-chief is a father who loves all his soldiers equally, and for that reason they are comrades among themselves.

In these two artificial groups, each individual is bound by libidinal ties, on the one hand, to the leader, and, on the other hand, to the other members of the group. Freud now thinks he is on the right track toward an explanation of the individual's lack of freedom in a group, the principal phenomenon in group psychology. He attributes to these emotional ties the alteration and limitation observed in each personality.

In developing the libidinal organization of groups, other examples of mutual relations between the object and the ego are considered—being in love and hypnosis. After these considerations, Freud feels in a position to construct the following formula for the libidinal constitution of groups: "A primary group is a number of individuals who have substituted one and the same object for their ego ideals and have consequently identified themselves with one another in their ego." This does not solve the riddle of the group, but simply shifts the question on to the riddle of hypnosis. Further effort leads to the matter of tracing the ontogenesis of the herd instinct.

From the first, there were two kinds of psychologies—that of the individual group member and that of the father or chief. Members were held by ties, but the chief was unhindered in any of his activities. His ego had no ties. He loved only himself or other people who served his needs. This is a familiar picture today. The members of a group stand in need of the illusion that they are equally and justly loved by their leader. But the leader himself need love no one else. He may be of a masterly nature, absolutely narcissistic, but self-confident and independent.

On the death of the primal father, a younger son or member replaces him. Here is the possible evidence of the transforming of group psychology into individual psychology. He depicts the primal father forcing his sons into sexual abstinence and consequently into the emotional ties with him and with one another. His sexual jealousy and intolerance forced them into group psychology. His successor emerges from this state to the condition of individual psychology. This derivation from the horde should help to explain, in group formations, the riddle of hypnosis and suggestion. The uncanniness of hypnosis suggests something old and familiar that has undergone repression. The hypnotist is thought to be in possession of a mysterious power which deprives the subject of his own will. This magic power is the same as that emanating from kings and chieftains, who are, because of it, dangerous to approach.

The derivation of the uncanny and coercive characteristics of groups, which are shown in their suggestion phenomena, are thus traced back to the primal horde. The leader is still the dreaded father and the group has a thirst for obedience. The father is the group ideal which governs the ego in the place of the ego ideal.